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CLINICAL PICTURE OF SEVERE GENERALIZED VIRAL INFECTION IN THE NEWBORN.[1]

By JOHN H. COLEBATCH.

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GENERALIZED infection of viral nature causing a fatal illness in early infancy was first reported fifty years ago (Ribbert, 1904; Jesionek and Kiolemenoglou, 1904). The disease as described then has been known as generalized visceral inclusion disease of infancy, or as generalized cytomegalic inclusion disease, or as generalized salivary-gland virus infection. Characteristic inclusion bodies are found in the nucleus and cytoplasm of the enlarged cells of various organs, most commonly the lungs, kidneys, liver and pancreas. Isolation of the virus causing this disease has not yet been successfully attempted. However, in 1941 the virus of *herpes simplex* was shown by Smith *et alii* to be responsible for a fatal encephalitis with similar intranuclear inclusion bodies developing in the neonatal period, and more recently there have appeared three overseas reports of fatal generalized visceral infection in the first week of life, in which the cause was definitely established as the virus of *herpes simplex* (Quilligan and Wilson, 1951; Zuelzer and Stulberg, 1952; Pugh, Newns and Dudgeon, 1954).

Neither generalized cytomegalic inclusion disease nor *herpes simplex* viræmia seems to have found a place in Australian reports until the past year. However, in August, 1953, the writer's interest was greatly stimulated by a newborn infant who appeared clinically to have died from a generalized viral infection. Jack and Williams (1955), with great perspicacity and flawless technique, at the post-mortem examination showed that the cause of death was generalized infection with the virus of *herpes simplex*. The striking similarity of the clinical pictures produced by these two apparently separate diseases prompted a study of the clinical features of cases reported in the literature and of similar cases encountered in Melbourne in recent years.

Generalized Cytomegalic Inclusion Disease.

Generalized cytomegalic inclusion disease may occur at any age, though its frequency and severity are greatest in early infancy. About 100 cases have been reported, a quarter of them in the neonatal period and five in still-born fœtuses. Some confusion arises from the fact that although most authorities consider that the virus responsible for this disease has not been identified, in some

[1] Based on a paper read at a meeting of the Pædiatric Society of Victoria on August 11, 1954.

cases reported as inclusion encephalitis a herpes-like virus was isolated (Malamud *et alii*, 1950). This emphasizes the similarity of cytomegalic inclusion disease to generalized *herpes simplex* infection; but it does not mean that the two conditions are caused by the same virus.

A benign inclusion disease of the salivary glands is a not infrequent finding in routine post-mortem examinations on infants in some localities (Farber and Wolbach, 1932; McCordock and Smith, 1934). When the disease occurs in generalized form, the organs that are likely to be affected include the lungs, kidneys, liver, pancreas, thyroid, brain, pituitary gland, spleen, bone marrow and alimentary tract. Although it follows from this list that the clinical manifestations can be protean, a fairly characteristic syndrome is usually presented when a newborn infant is affected.

The patient is apt to be a premature or debilitated infant with symptoms present at or within a few days of birth. Feeding difficulties, lethargy and jaundice develop early. Respiratory distress and cyanosis are common findings. Hæmorrhagic phenomena usually appear, such as bleeding from the cord, injection sites, bladder, bowel, stomach *et cetera*, together with petechiæ, thrombocytopenia and often anæmia, with numerous erythroblasts in the blood. The liver and spleen are usually considerably enlarged. In some cases encephalitis develops, and X-ray examination may show small intracerebral calcifications similar to those in toxoplasmosis. Vomiting is a common symptom, sometimes followed by diarrhœa. Involvement of the kidneys may make it possible to diagnose the condition during life by the finding in the urinary sediment of tubular epithelial cells containing cytoplasmic and even intranuclear inclusion bodies (Fetterman, 1952; Mercer *et alii*, 1953).

The clinical picture of generalized cytomegalic inclusion disease is determined to a large extent by the age of the patient. In the reports of the disease in stillborn fœtuses, the kidneys have been involved in all five cases, the lungs in four, the liver in three and the adrenals in at least one. Cases in which the condition develops in the first day or two of life usually present a clinical picture closely resembling that of erythroblastosis due to Rh incompatibility. A hæmorrhagic diathesis with thrombocytopenia and hepato-splenomegaly is a common presenting syndrome in the first week of life. Later, especially after the first month, involvement of the lungs is more likely to predominate, or sometimes severe diarrhœa. Reports of the coexistence of pertussis or of fibrocystic disease of the pancreas in a number of affected children aged over three months raise doubts as to the clinical importance of the viral infection in them, while in affected adults it is usually obvious that some condition other than the inclusion disease has been the cause of death. Several comprehensive reviews of the literature have been published (Wyatt *et alii*, 1950; Smith and Vellios, 1950; Kidder, 1952; Bacala and Burke, 1953).

In newborn infants death commonly occurs within a few days but when the onset has been later than the first twenty-four hours of life the course may be longer and even recovery may result (Gallagher, 1952). Information is too scanty to permit of assessment of the prognosis in this disease; but the pathological evidence would lead one to expect, in an infant who survives, sequelæ such as cirrhosis of the liver, nephrosclerosis, cystic disease of the lungs and pancreas, mental retardation and hydrocephalus.

In August, 1954, Quinn of Brisbane presented two cases of generalized cytomegalic inclusion disease, one in the neonatal period, the other discovered in an infant aged six months. Also, two cases in later infancy have recently been recognized at autopsy in Adelaide (Fowler, 1954). The following clinical history of a patient from Sydney who survived and is now under the writer's care is probably an example of this disease, though the diagnosis lacks histological confirmation.

CASE I.—A., a female patient, had jaundice, cyanosis and purpura at birth. Some months before conception, her mother, a *primigravida*, had had a mild illness said to resemble encephalitis. She vomited throughout pregnancy, yet gained weight excessively. Miscarriage was threatened at six weeks and at six months. At eight months the mother was admitted to hospital with toxæmia of pregnancy and severe hydramnios. She was delivered ten days before term, after a normal labour.

At birth the infant weighed four and a half pounds and had a large liver and spleen, in addition to purpura, cyanosis and jaundice. There was no reaction to the Coombs test. Thought to be dying from severe hæmorrhage, she was given a blood transfusion on the third day. Two days later intracranial hæmorrhage was suspected. Gradually her condition improved; but when she was discharged home at the age of six weeks she still showed jaundice and a tendency to vomiting and diarrhœa, aggravated by cow's milk. Cough and fever were present intermittently, and at the age of three months the patient developed pneumonia, followed by a severe flare-up of the vomiting and diarrhœa. Dr. C. W. G. Lee, who examined her then, described her as almost moribund. The liver and spleen were still enlarged, persistent neutrophilia was present, and at one stage the urine contained albumin, red blood cells and numerous casts. No inclusion bodies were found in the urine. Normal results were obtained from many other investigations, including examination of the cerebro-spinal fluid, a Wassermann test of the blood, bone marrow puncture, a Mantoux test, and cultural examination of the stool. From this stage there was a very slow but steady improvement, punctuated by upper respiratory tract infections and another attack of pneumonia at the age of nine months.

When examined by the writer at the age of fifteen months, the patient was several pounds under weight, unable to crawl, stand or walk and partially deaf, with enlargement of the liver and spleen. From numerous investigations the only abnormal finding was a high serum albumin level (5·5 grammes per 100 millilitres) with an albumin-globulin ratio of 3:1. *Herpes simplex* complement-fixing antibody was absent from the child's serum, but it was present in the serum of the mother, who was known to have recurrent *herpes labialis*. At the age of two years the child had been able to walk for several weeks and the spleen was impalpable; but she still appeared mentally retarded and had not commenced to speak.

The diagnosis of generalized cytomegalic inclusion disease in this case can be no more than a presumptive one. However, the clinical picture conforms so closely to the pattern of this disease in the newborn as to be worth placing on record. The syndrome of jaundice, hepato-splenomegaly and purpura, present at or within a day or two of birth, with a negative response to the Coombs test, is the usual mode of presentation of this disease when the infant is affected at birth. The cyanosis and other respiratory symptoms, the diarrhœal bouts and early intolerance to cow's milk, and the subsequent mental retardation are all in keeping with this clinical diagnosis. Failure to find inclusion bodies in the urine as late as the fourth month is of little value in excluding the condition.

Herpes Simplex Viræmia.

Hass in 1935 suggested that the virus of *herpes simplex* could pass internally to cause a generalized infection. In the liver and adrenals of a premature infant who died in the second week, he found intranuclear inclusion bodies characteristic of *herpes simplex* infection. Support for his hypothesis has been provided since 1940 by the isolation of this virus from the brain in at least seven cases of "inclusion encephalitis" (two in newborn infants) and from skin vesicles in a newborn infant who survived acute herpetic dermatitis with encephalitis. In these cases there was no evidence of visceral lesions, so the presence of a *herpes simplex* viræmia is doubtful, as it is possible that the virus may reach the brain by routes other than the blood-stream. However, since 1951 there have been reported four neonatal cases (including our Australian one) in which the virus of *herpes simplex* has been isolated from the liver; in one of these, it was also isolated from skin lesions; in two of them there was evidence of encephalitis as well as visceral disease. The existence of *herpes simplex* viræmia as an entity must therefore be accepted.

A pathologically distinct form of hepatitis with multiple foci of necrosis and the characteristic intranuclear inclusion bodies has been a prominent feature common to these four proven cases of neonatal *herpes simplex* viræmia. There are reports in the literature of at least another seven

neonatal cases in which the same pathological features justified a presumptive diagnosis of *herpes simplex* viræmia, but isolation of the virus was not attempted (Hass, 1935; Zuelzer and Stulberg, 1952; France and Wilmers, 1953). In this paper four additional cases of this type are described, together with a fifth in which the clinical and some of the histological features were of a similar nature. Zuelzer and Stulberg (1952) have also described three fatal cases in later infancy, in which the same characteristic hepatitis was present.

Unlike generalized cytomegalic inclusion disease, *herpes simplex* viræmia has never been reported as an intrauterine infection. The earliest onset of relevant symptoms has been on the fourth day of life, and in 12 of the 15 cases under review the onset fell between the fourth and seventh days. Death occurred most commonly on the eighth or ninth day of life. The duration of the illness was three to five days in most cases, but in one it was thirty-five days. By contrast, it is to be noted that in newborns shown to have *herpes simplex* encephalitis without any visceral involvement, the onset of cerebral symptoms has been later (usually in the second or third week), and the duration of the illness has been longer, ending in two cases with the survival of the patient. *Herpes simplex* viræmia may also occur in later infancy, as indicated by the three patients under the age of two years reported by Zuelzer and Stulberg (1952). In these three cases, an herpetic or aphthous stomatitis was present at the onset and the characteristic hepatitis was found at autopsy. In two of them a coexistent disease—pertussis in one, Hirschsprung's disease in the other—was probably the chief cause of death. This led Zuelzer and Stulberg to suggest that viræmia and visceral involvement might be commoner in primary herpetic stomatitis than had been generally thought.

Infection with the *herpes simplex* virus may be contracted by the infant during its passage from the vagina of a mother suffering from genital herpes, or later by droplet infection from, or physical contact with, the mother, a nurse or any other attendant who is carrying the virus in the mouth. The infant's oro-pharynx, skin and conjunctiva are thus the commonest portals of entry, and this can also be inferred from the pathological findings in at least 12 of the 18 cases reviewed. Lest the importance of the attendants as a frequent source of infection be overlooked, it is worth noting that in only three out of 15 neonatal cases has the presence of genital or labial herpes in the mother been detected.

The mode of infection and portal of entry, together with the age of the patient, will influence the distribution of pathological lesions in the body and the clinical picture that results. It is not the purpose of this paper to give an account of the pathological findings, which have been well described by other writers (Quilligan and Wilson, 1951; Zuelzer and Stulberg, 1952; Jack and Williams, 1955). However, it is pertinent to mention that at autopsy in 18 cases, the organs most often reported to be involved have been the liver (18 cases), the oro-pharynx, œsophagus and/or stomach (11 cases), the adrenal cortex (eight cases), the spleen, lungs and brain (five cases each), the kidney and bone marrow (four cases each), the lymph glands, skin and conjunctiva.

The following clinical description is based on the reports of the 15 cases previously mentioned, which can be regarded with reasonable certainty as examples of *herpes simplex* viræmia in the newborn. Reference will also be made to the clinical data of the three additional cases in older infants and to Case VII of the present paper which follows the same clinical pattern. As might be expected, the picture in the first week or two of life in most respects closely resembles that of generalized cytomegalic inclusion disease.

The patient is apt to be a premature or debilitated infant. Seven of the 15 neonatal patients under review were premature; the average birth weight of the eight full-term infants was only six pounds seven ounces, and several of them had atelectasis. No symptoms related to *herpes simplex* infection are to be expected for the first three or more days of life. Listlessness and difficulty with feedings, perhaps with dyspnœa or choking attacks, usher in the illness, except in those cases (four out of 15 reported) in which these symptoms are preceded by herpetic stomatitis, conjunctivitis or dermatitis. The following are the commonest early findings: respiratory distress and chest retraction, often with attacks of cyanosis; a pathological degree of jaundice, steadily deepening; and an unstable temperature, which may be high, low or unduly fluctuating. A day or so later, tachycardia may be noted, vomiting often develops, and sometimes the stools become loose, with blood and mucus present. By this time the liver is frequently enlarged, and occasionally the spleen also. The condition of the infant then deteriorates rapidly. At least half the patients develop an hæmorhagic diathesis in increasing severity, the skin, bowel, lungs and umbilicus being the usual sites of bleeding, with consequent anæmia. In a few cases evidence of encephalitis develops, generally with terminal convulsions. The temperature may rise precipitously, or more commonly it falls to levels three or four degrees below normal. In a matter of hours, increasing collapse, respiratory distress and cyanosis, perhaps with terminal brachypnœa, lead to death. In the three cases in which the illness began in the second week of life the symptomatology differed, in that jaundice was absent and hæmorrhagic manifestations were minimal.

Very few investigations have been carried out in the cases under review. Albuminuria, which has been recorded, is a normal finding in many infants in the first week of life. No examination of the urinary deposit for inclusion bodies in epithelial cells appears to have been undertaken. Thrombocytopenia was present in two cases, but in most of them it may not have been looked for. The plasma prothrombin level was stated to have been below normal in two other cases with hæmorrhagic manifestations. The leucocyte count, recorded in six cases, has been normal apart from slight lymphocytosis in one and neutrophilia in another. An interesting feature is that the cephalin flocculation test, carried out in only two cases, has produced a strongly positive result in both. Virological studies are outside the scope of this paper, but they are obviously of the greatest importance. It is hoped that clinicians in future will be able to suspect the presence of generalized *herpes simplex* infection in time to submit specimens of blood, serum, urine and oropharyngeal secretions to a virus laboratory.

CASE II.—B., a female patient, was born on July 28, 1953. There was a family history of allergic disorders and of duodenal ulcer. The mother, a *primigravida* under the care of Dr. George Simpson, suffered from *pruritus vulvæ* in the last four months of pregnancy. Fifteen days before delivery she also had a febrile attack of "influenza" with headache, lassitude and sore throat, which subsided without treatment, apart from a residual cough and coryza. She had an uneventful delivery at term, the presentation, labour and placenta being normal. The Wassermann test on the mother's serum produced a negative result.

At birth the length of the infant was 19 inches and the weight six pounds ten ounces. She revived normally and was progressing well when transferred with her mother to the convalescent wing on the second day. On the sixth day of life she was lethargic, and dusky in colour after a feeding, vomiting a little and passing a few loose stools. The temperature rose that night to 102·4° F. and a few rhonchi were heard over the lungs. When she was examined by the writer next morning her pulse rate was 180 per minute and her respirations numbered 40 to 56 per minute; slight jaundice was present; there was chest retraction on inspiration; weight loss was excessive (13% of the birth weight). The picture suggested atelectasis of minor degree with dehydration. Increased fluids were ordered, together with oxygen and antibiotics. During the next two and a half days there was no cyanosis or vomiting, the increased fluids (20 to 24 ounces *per diem*) were taken well, and the pulse and respiration rates were within normal limits; but some looseness of the stools persisted and the maximum temperature each day was between 100·4° F. and 102° F.

At the end of the ninth day part of the feedings was refused, the pulse rate rose to 190 per minute and the respirations to 76 per minute, and minimal umbilical infection was noted. By the next morning feeding by gavage was necessary, and vomiting and diarrhœa of minor degree were

present. Dr. T. G. Maddison, who observed the infant carefully over the next two days, noted that the liver was palpable one finger's breadth below the costal margin, and that the heart was not clinically enlarged despite a pulse rate of 196 per minute. "Aureomycin" was prescribed. Early on the eleventh day the temperature, which had been elevated for four days, dropped below normal, and thereafter it continued to fall. Pallor and irritability were present, and later in the day a state of severe shock developed, the pulse rate being only 98 per minute and the respirations 40 per minute. Blood-stained gastric residue was aspirated, petechiæ appeared on the abdomen, the hæmoglobin value was 8·7 grammes per 100 millilitres (60%), and the liver was then enlarged and palpable three and a half fingers' breadth below the costal margin. A transfusion of 180 millilitres of blood was given. Shortly afterwards, a blood examination revealed a hæmoglobin value of 17·4 grammes per 100 millilitres (120%) and a leucocyte count of 14,400 per cubic millimetre with a normal differential count, and the platelets numbered less than 50,000 per cubic millimetre. The infant remained gravely ill in spite of ACTH, "Aureomycin" given intravenously, and the blood transfusion. Hypothermia, rapidly increasing hæmorrhage from the stomach, bowel and elsewhere, and convulsions led to her death at the commencement of the twelfth day. From these clinical data the impression was recorded that this infant had died from an acute generalized infection, probably viral, involving the liver, lungs, heart and bone marrow.

Pathological and virological studies *post mortem* have been reported in detail elsewhere (Jack and Williams, 1955). In brief, the main pathological findings were as follows: extensive atelectasis; widespread petechial hæmorrhages; numerous foci of necrosis with intranuclear inclusion bodies in the liver, the adrenal cortex and the œsophagus, and some inclusion bodies in mononuclear cells in the alveoli of the lungs. *Herpes simplex* virus was isolated from the liver. Examination of the blood of both parents revealed *herpes simplex* complement-fixing antibody in the father (who had had no contact with the infant), but none in the mother.

This is the first Australian case of *herpes simplex* viræmia that has been confirmed by isolation of the virus. The full clinical history has been recorded because it provides such an excellent example of the pattern of this disease as to be of assistance to clinicians who may be faced with similar diagnostic problems in the future, and because most of the case reports in the literature contain only scanty clinical notes. The viral antibody tests on the parents are of particular significance. In the first place, the absence of antibody from the mother's serum indicates that she was not a sufferer from or a carrier of the virus, so that her *pruritis vulvæ* and febrile "influenza" during pregnancy could not have been herpetic in nature, as some writers are prone to assume. Secondly, as neither the mother nor the father could have infected the patient, the source of the virus must have been an attendant. During the infant's four days in the convalescent wing of the hospital, some of the attendants were suffering from *herpes labialis*, and as the usual incubation period for primary herpetic infection is three or four days, one of these attendants may well have been the source of the infection in this case.

In the next four cases, with the finding of characteristic herpetic hepatitis the diagnosis of *herpes simplex* viræmia appears justified though its proof by isolation of the virus was not attempted.

CASE III.—C., a female infant, was the first-born of heterozygous twins in the mother's eighth pregnancy. Ante-natal care was minimal, but pregnancy and delivery appeared to have been normal. The infant weighed seven pounds two ounces at birth on September 26, 1952, and she progressed normally till the fifth day, when the mother insisted on going home with both twins. From the sixth day the patient took her feedings poorly and later seemed weak and in pain whenever she was handled. She was fed at 3 a.m. on the ninth day. Three hours later she was found dead in bed.

At the post-mortem examination by Dr. Keith Bowden the main findings were intranuclear inclusion bodies in necrotic areas in the liver, and widespread hæmorrhage into lungs and liver. It is instructive to note that the second twin, a male, whose birth weight was eight pounds five ounces, vomited some "coffee grounds" material on the second day of life, but that thereafter his progress was entirely normal.

CASE IV.—D., a male infant, weighed three pounds twelve ounces when born on May 27, 1953. The mother was a *primigravida* with no evidence or history of *herpes simplex* infection. Severe toxæmia of pregnancy led to delivery by Cæsarean section in a private hospital after thirty-four and a half weeks' gestation. The infant had attacks of cyanosis two and four hours after delivery, and duskiness intermittently thereafter. At the end of the first day, on his admission to the Royal Children's Hospital under the care of Dr. L. P. Wait, he had signs of atelectasis and physiological jaundice with a palpable liver. He responded to treatment, including the administration of oxygen and vitamin *K*.

Early on the fifth day the temperature was recorded as 101° F. on one occasion, but it was normal for the next few days. On the sixth day the infant vomited once and a generalized erythemato-macular rash appeared, but within twenty-four hours it had vanished. Up to this stage, weight records indicated that feedings had been taken well. On the eighth day the stools increased in number, and the temperature rose to a plateau level of 100° to 101·4° F. Next day he was lethargic and unable to take all his feedings, and later became cyanosed with increased pulse and respiration rates and persistent jaundice. By the tenth day he was gravely ill with vomiting, failure to absorb feedings given by gavage tube, cyanosis with irregular shallow respirations, and a temperature that was below normal and still falling. He survived until halfway through his eleventh day.

At the autopsy, Dr. A. Williams found focal necrosis with intranuclear inclusion bodies in the liver and adrenal cortex, together with pulmonary atelectasis.

CASE V.—E., a female infant, was born on July 31, 1950, in a private hospital. There is no record of any abnormality in the mother's pregnancy or labour. The infant weighed six pounds seven ounces at birth, and her progress was described as normal when she was discharged home on the ninth day. A few days later she began to suck poorly and to refuse part of her feedings. When seventeen days old she was admitted to the Queen Victoria Hospital under the care of Dr. Gladys Hallows. There were a number of vesicular and what seemed to be purulent lesions on the skin of the right hand, the left leg and the back, a small ulcer on the palate and a palpable spleen. The condition of the patient steadily deteriorated. The temperature had been 96° F. on her admission to hospital, but it rose to 107° F. shortly before her death on the nineteenth day. At the post-mortem examination Dr. Elsie Abrahams found characteristic lesions with intranuclear inclusion bodies in the liver.

CASE VI.—F., a male infant, was the first of homozygous twins born in the Queen Victoria Hospital on May 31, 1954, to a multiparous mother. The infant was delivered by the breech nine weeks before term, but pregnancy and labour were otherwise normal. His birth weight was three pounds 13 ounces. He was cyanosed on delivery but revived well, and despite œdema he responded satisfactorily to the routine care for a premature infant.

On the fifth day he still seemed well, but the temperature rose to 101° F. and the jaundice was deepening. He vomited twice on the following day. Dr. Kate Campbell, under whose care he was placed, reduced the size of his feedings and for the next two days he was definitely better and afebrile. On the ninth day he was pale and icteric, his temperature fluctuating between 96·6° and 100·6° F. Despite intensive therapy, including the administration of broad-spectrum antibiotics, he developed diarrhœa, respiratory distress and cyanotic attacks, and he died on the tenth day. At the post-mortem examination Dr. Elsie Abrahams found characteristic hepatic lesions with intranuclear inclusion bodies.

The second twin, a male, weighing three pounds 12 ounces, died on the second day from pulmonary atelectasis and intracranial hæmorrhage. No inclusion bodies were found at autopsy.

The clinical features of these four cases provide support for the view that the pathological findings are those of visceral *herpes simplex* infection. In particular, the insidious onset, the unstable temperature, the respiratory symptoms and the rapid terminal decline are characteristic of the clinical picture as shown in the four reported cases in which the virus has been isolated. Case V illustrates the occurrence of superficial herpetic infection as a precursor to the generalized visceral infection. Herpetic infection of the skin or of the mouth has been present in four of the 15 neonatal cases, and in all three of the cases reported in later infancy. Cases II and IV of this series provided the material for the paper on hepatic necrosis by Jack and Williams (1955).

CASE VII.—G., a female infant, was born at term after a normal delivery on April 24, 1952. The birth weight was five pounds 12 ounces. For five days her progress was normal, physiological jaundice developing on the third day.

On the sixth day the jaundice was increasing, anorexia was noted and the colour was ashen at times. On the seventh day the infant's colour was worse, and she had grunting respirations and inability to suck. She was admitted to the Children's Hospital under the care of Dr. R. Southby. Examination of the patient revealed jaundice, a pulse rate of 170 per minute, respiratory distress, enlargement of the liver to two and a half fingers' breadth below the costal margin, a palpable spleen and a hæmoglobin level of 17·7 grammes per 100 millilitres (112%). On the following day there was increasing dyspnœa with cyanosis and some crepitations over both lungs, the temperature rose to 100·8° F., the spleen was enlarged to two fingers' breadth below the costal margin, and skin bleeding developed particularly from an intravenous wound. Infection of the umbilicus was also apparent, and *Staphylococcus aureus* was subsequently grown on culture from it. Blood examination showed a hæmoglobin level of 13·9 grammes per 100 millilitres (94%), nucleated red cells numbering three per 100 leucocytes, a leucocytosis of 47,000 per cubic millimetre, of which 64% were neutrophile leucocytes and 34% were lymphocytes, and a "greatly reduced" number of platelets. Intensive therapy, including the administration of antibiotics, vitamin *K* and blood transfusion, failed to control the increasing hæmorrhage and cyanosis, and death occurred on the ninth day. At the post-mortem examination by Dr. J. W. Perry, the main lesions were massive focal necrosis of the liver, renal tubular damage, patchy myocarditis, and pulmonary congestion and collapse with early bronchopneumonia. However, no inclusion bodies could be found.

This case presents an interesting problem in diagnosis. The clinical features follow very closely indeed the pattern now known to be produced by *herpes simplex* viræmia. The umbilical infection and leucocytosis are not part of this pattern, but they could have been the result of a coexistent secondary infection. However, in the light of our present knowledge, the absence of inclusion bodies from the liver and other viscera would seem sufficient basis for excluding *herpes simplex* viræmia. If this is so, this case merely serves to illustrate that, at least in the first week of life, it may not be possible either to establish or to exclude a diagnosis of *herpes simplex* viræmia before death.

Discussion.

Generalized cytomegalic inclusion disease and *herpes simplex* viræmia are two apparently distinct generalized inclusion diseases due to viruses which can cause a rapidly fatal illness in the newborn, and which may affect older subjects in a less dramatic way. Though they have many pathological features in common, the former disease, compared with *herpes simplex* viræmia, is far more prone to affect the kidneys, pancreas, thyroid and salivary glands, its lesions show less necrosis, and above all the cells containing inclusion bodies show a much greater degree of enlargement, for which reason Goodpasture and Talbot applied the term "cytomegalia". In *herpes simplex* viræmia the liver, adrenal cortex, oro-pharynx and œsophagus are most often involved, necrosis is a prominent feature, and huge inclusion-bearing cells are not seen. At autopsy, therefore, the pathologist is in a favourable position to differentiate between these diseases.

The clinician has a more difficult problem. In the first place, it is not easy to decide whether an infant is in fact suffering from a generalized viral infection, as Case VII illustrates. This possibility may be suspected in any severe or steadily progressive illness in which alimentary, respiratory, hæmorrhagic or cerebral symptoms appear in the first week or so of life, particularly if the liver or spleen is enlarged and the leucocyte count is normal. The temperature may be elevated, but more often it is not. Intranuclear inclusion bodies may be found in the urinary sediment in at least one of these inclusion diseases. Eventually a septicæmic syndrome is likely to develop. However, the very multiplicity of the symptoms may bewilder and confound the clinician. A further difficulty is provided by the great variability that the age of the patient produces in the pattern of pathological lesions and clinical features. In generalized cytomegalic inclusion disease this is strikingly illustrated. The clinical pictures produced by this infection in the fœtus, in the first two days of life, in the subsequent neonatal period, in later childhood and in adult life differ so widely that the clinician can scarcely recognize them as having a common ætiology.

Secondly, in the newborn the clinical pictures of these two generalized viral infections resemble each other so strongly that their differentiation during life may be impossible. In both diseases the liver and lungs are usually involved, frequently also the kidneys, adrenal cortex, alimentary tract, spleen and bone marrow, and in some cases the brain. Common to both diseases, therefore, are syndromes of respiratory disorder or of liver dysfunction, hæmorrhagic states, diarrhœa and vomiting and encephalitis. Surprisingly, no clinical study of these diseases and their differentiation has appeared in the literature. Perhaps this is because generalized cytomegalic inclusion disease has been "largely a pathologist's disease", while *herpes simplex* viræmia has only recently been established as an entity.

One useful guide in distinguishing between these two diseases is the time of onset of the illness. There is no report of *herpes simplex* viræmia commencing before the fourth day of life. Generalized cytomegalic inclusion disease, on the other hand, frequently appears in the first forty-eight hours and may even be found in stillborn fœtuses. In the later neonatal period, when alimentary, respiratory or hæmorrhagic symptoms may predominate, the differential diagnosis is more difficult, but the following points provide some help. *Herpes simplex* lesions in the skin, mouth or conjunctiva furnish presumptive evidence that associated systemic lesions are due to the same virus. Thick, blood-stained, mucopurulent secretions from the pharynx may indicate herpetic œsophagitis. In generalized cytomegalic inclusion disease, enlargement of the spleen is the rule, and fluctuations of temperature are unusual, while in both respects the reverse holds for *herpes simplex* viræmia. Finally, small foci of intracranial calcification may follow an inclusion encephalitis of cytomegalic type, but they have not been recorded in cases due to *herpes simplex* infection.

Several authors have postulated a special susceptibility of embryonic tissues to explain the severe generalized disease produced in the newborn by each of these infections. Analogous experiences with *herpes simplex* virus in chick embryos and with salivary-gland virus in guineapigs, together with the prematurity of many of the human patients, justify this hypothesis. Others have suggested that the non-immunity of the mother is the vital factor, as might be illustrated by Case II of the present series. While both these predisposing factors are probably important, they do not provide a wholly satisfactory explanation, as a number of the patients have been full-term infants a week or more old, and in at least one proven case of *herpes simplex* viræmia (Zuelzer and Stulberg, 1952, Case I), the mother's serum contained specific antibodies in high titre at the time of the infant's illness.

It may well be significant, therefore, that the present study of the reports of 15 cases of *herpes simplex* viræmia showed in every case that prior to the onset of the illness there was evidence of debilitating factors calculated to lower the defensive powers of the infant. Maternal toxæmia and hypertension were present in four cases, and febrile infection in the last fortnight of pregnancy in three cases. Delivery by Cæsarean section was reported in two cases, multiparity in five, immaturity (judged by gestational age) in nine, and low birth weight of less than six and a half pounds in ten. Atelectasis or cyanotic attacks on the first day of life were mentioned in four cases. In all 15 cases at least one of these preexisting causes of debility was present, and two or more of them were recorded in each of 11 out of 13 adequately documented cases. Considering the recurrence of *herpes labialis* in later life that takes place in response to minimal debilitating influences, it seems probable that the severity of neo-natal herpetic infections is to a significant degree due to the antecedent debilitating factors mentioned.

Summary.

1. The literature is reviewed relating to the clinical features and problems of two generalized viral infections that are usually fatal in the newborn—generalized cytomegalic inclusion disease and *herpes simplex* viræmia.

2. One probable case of generalized cytomegalic inclusion disease is described.

3. An analysis is presented of the clinical data from 15 case histories of *herpes simplex* viræmia, which has only recently been established as an entity.

4. Five cases considered to be examples of *herpes simplex* viræmia are described. In one of these the virus was isolated for the first time in Australia (Jack and Williams, 1955).

5. The differential diagnosis of these two clinically similar but apparently separate virus infections is discussed for the first time.

6. Clinical data are presented to indicate that antecedent debilitating factors may play a significant role in these severe neonatal virus infections.

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SOME OBSERVATIONS ON THE COMPLEMENT-FIXATION TEST FOR THE PSITTACOSIS-LYMPHOGRANULOMA VENEREUM GROUP OF VIRUSES.

By D. Surrey Dane,[1]

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A method of preparing a complement-fixing antigen for the psittacosis-lymphogranuloma group of viruses is described below, and certain factors in the diagnosis of psittacosis by means of the complement-fixation test are considered.

Materials and Methods.

The complement-fixation test was performed in the manner described elsewhere (Dane, 1955). The preparation of antigens for the psittacosis-lymphogranuloma group of viruses is described in the text. The soluble murine typhus antigen used was purchased from Lederle Laboratories.

Results.

Psittacosis Lymphogranuloma Group Antigens.

The following method of preparing group antigen has been used in this laboratory for the past year with satisfactory results.

Embryonated hens' eggs, which have been incubated for thirteen days, are inoculated via the yolk-sac with a suspension of enzootic abortion of ewes virus of sufficient strength to kill the majority of the embryos on the fifth day. Eggs are candled daily, and those dead before the fourth day are discarded. The yolk-sacs of eggs dead on the fourth, fifth and sixth days are harvested. Smears of yolk-sac are stained overnight with Giemsa's stain to check that good virus growth has occurred, and the yolk is cultivated on blood agar to ensure that there is no bacterial contamination. Suitable yolk-sacs are shaken with 10 millilitres per yolk-sac of 50/M phosphate buffer at pH 7·2 and allowed to stand overnight for extraction at +4° C. The suspension is then lightly centrifuged to deposit the larger particles of yolk-sac, and the supernatant is centrifuged at 8500 revolutions per minute for one hour, to deposit the virus. The virus is resuspended in normal saline with the aid of a syringe and wide-bore needle, and the suspension is then made up to its original volume. This partially purified suspension is placed in a boiling water-bath for twenty minutes, and then after having been cooled is shaken with two volumes of anæsthetic ether and left at room temperature. It is shaken thoroughly every few hours during the daytime for four days, and then the ether layer is drawn off and evaporated over a volume of calcium magnesium saline (Mayer, 1946) equivalent to that of the original suspension. "Merthiolate", one part in 10,000, is added as a preservative, and the antigen is stored at refrigerator temperature. Control antigens are prepared in a similar way from normal eggs of the same age.

The preparation of the boiled suspension of virus before the ether extract is made is similar to that described by Monsur and Barwell (1951). The heated ether extract antigen gave comparable results (see Table I) to antigens prepared by their method from two strains of psittacosis virus recently isolated in this laboratory, when tested against 14 "positive" human sera found in a survey of normal people described elsewhere (Dane, 1955). The extraction of group complement-fixing antigen from yolk-sac suspensions of lymphogranuloma virus with anæsthetic ether has been described by Hilleman and Nigg (1946). It was found that ether extracts prepared from unheated suspensions of enzootic abortion of ewes or psittacosis virus gave irregular results. This was especially true with some immune pigeon sera, which failed to react or to react fully with these antigens. Heating before the ether extract was made produced an antigen which reacted fully with all known "positive" sera. No reactions with the heated ether extract control antigens have been found in tests on over 600 normal human sera, and on 150 sera from

[1] Assisted by a grant from the National Health and Medical Research Council.

TABLE I.
Comparison of Antigens.

Antigens.		Serial Numbers of Sera.													
Method of Preparation.	Virus.	500	502	505	507	508	509	512	514	517	518	520	528	530	532
Ether extract of heated suspension.	Enzootic abortion of ewes ..	40[1]	20	80	20	5	40	40	80	5	20	80	40	10	5
Heated suspension.	Psittacosis AR	40	20	40	20	5	40	20	80	5	20	80	40	5	10
	Psittacosis HP ..	20	10	40	10	<5	20	10	40	5	10	40	20	<5	5

[1] Reciprocal of serum titre.

patients suffering from pneumonia and/or from pyrexia of unknown origin (Beech, 1954, unpublished). Serum was tested at an initial dilution of one in five and the control antigen was used at twice the optimum strength of the enzootic abortion of ewes antigen. The quantity of antigen produced by the method described above is satisfactory, and the antigen is not anti-complementary in useful dilutions. A heated ether extract enzootic abortion of ewes antigen prepared from six eggs gave 9·6 litres of complement-fixing antigen at optimal dilution, and anti-complementary activity was not detectable until 16 times this strength was used. The use of enzootic abortion of ewes virus for the production of group antigen seems preferable to the use of psittacosis virus, because the former is safer to handle and grows to a higher titre in the yolk-sac.

TABLE II.
Routine Serological Tests.

Serial Number of Patient.	Weeks After Onset of Illness.	Complement Fixation Test.			Proteus OX19 Agglutination.	Diagnosis.
		Psittacosis Group.	"Q" Fever.	Typhus.		
97	5	320[1]	<5	—	—	Psittacosis.
	7	640	<5	80	<5	
	18	640	—[2]	—	—	
	22	320	—	20	<5	
113	1	5	<5	<5	320	Murine typhus.
	17	<5	<5	40	—	
168	4	640	<5	—	640	Murine typhus.
	6	640	—	10	—	
	10	160	<5	160	—	
176	2	10	<5	—	—	"Q" fever.
	4	80	1280	—	—	
173	1	20	20	—	—	"Q" fever.
	3	20	1280	—	—	
178	1	<5	<5	—	—	"Q" fever.
	2	<5	<5	—	—	
	3	5	320	—	—	
209	1	<5	<5	—	—	"Q" fever.
	2	<5	>640	—	—	
87	2	20	40	—	—	"Q" fever.
	4	40	320	—	—	
	7	40	320	—	—	

[1] Reciprocal of serum titre.
[2] Not tested.

The Serological Diagnosis of Psittacosis.

The complement-fixation test with the use of a group antigen is the serological test most commonly employed in the diagnosis of psittacosis, and it is the only one which will be considered here. In the past the demonstration of at least a fourfold rise in titre during the course of an illness, together with a convalescent titre of more than 1 in 40, was considered necessary for making a serological diagnosis. If only convalescent serum was available for testing, a titre of more than 1 in 40 was considered suggestive. Now that antibiotics are almost invariably used to treat suspected psittacosis, and now that complement-fixing antibodies are known to be widely distributed in the general population, some reconsideration of these diagnostic criteria is necessary. Recently French and his associates (1954) and Sigel (1953) have drawn attention to the types of antibody response that may be met with in psittacosis treated with antibiotics. Complement-fixing antibody titres do not always rise to a high level, and a rapid fall in titre may occur during convalescence. Also, the appearance of complement-fixing antibodies in the blood may be considerably delayed. The fact that titres do not necessarily rise to a high level is important, because of the prevalence of low titre antibodies in normal people (Gerloff and Lackman, 1954; Dane, 1955). An early convalescent specimen of serum may have a titre of only 1 in 10 or 1 in 20; but such titres are comparatively common findings in normal individuals. The demonstration of a rising titre during an illness is therefore even more important than it was formerly. However, owing to the rapid fall in titre which may follow antibiotic treatment, it is no longer possible to distinguish a specific rise from an anamnestic rise, for both may be brief. As might be expected from the fact that psittacosis-lymphogranuloma group virus antibodies can be detected in serum from 22% of the normal population of South Australia (Dane, 1955), anamnestic rises are a common occurrence in other infections. Table II shows the psittacosis-lymphogranuloma group titres found in the patients who were diagnosed as having psittacosis, "Q" fever or murine typhus at this Institute during the twelve months from June, 1953, to May, 1954. In one case of "Q" fever (Case 87) there was a twofold rise in psittacosis-lymphogranuloma group antibody titre, and in another (Case 176) there was an eightfold rise. In a case of murine typhus (Case 168), six weeks after the onset of the disease the patient had a psitttacosis-lymphogranuloma group titre of 1 in 640, and a titre to soluble typhus antigen of 1 in 10. Four weeks later the psittacosis-lymphogranuloma group titre had dropped to 1 in 160, and the titre to typhus antigen had risen to 1 in 160. The only case of psittacosis (Case 97) was that of a man, aged fifty years, who had nursed a sick budgerigar in his bedroom two weeks before the onset of his illness. In 1943 he had suffered from typhus in Siberia, and this accounts for the presence of complement-fixing antibodies to typhus antigen in his serum.

In this small series of cases there are two, Cases 176 and 168, in which a tentative diagnosis of infection with a virus of the psittacosis-lymphogranuloma group might have been made on serological grounds if more certain evidence of another infection had not been found.

Summary and Conclusions.

1. A method of preparing an antigen for the psittacosis-lymphogranuloma group complement-fixing test is described. This antigen gives satisfactory results and is comparatively safe, simple and economical to prepare.

2. Some of the difficulties which may be encountered in the serological diagnosis of psittacosis are considered.

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THE EFFECTS ON BLOOD DONORS OF REGULAR REMOVAL OF BLOOD.

By FLORENCE DAVIS.[1]

From the New South Wales Red Cross Blood Transfusion Service, Sydney.

A BLOOD transfusion service depends largely on regular donors for maintaining the supply of blood to hospitals. This fact raises an important question: Is a blood transfusion service justified in asking donors to give 400 to 500 millilitres of blood at regular three-monthly intervals? The service must clearly be satisfied that the regular removal of such an amount of blood has no injurious effect on the health of the donor.

A further reason for attempting to obtain scientific evidence on this question is a widespread impression in the community, and indeed among many doctors, that blood donation is injurious. No transfusion service would ever take blood from a donor who showed any obvious sign of ill-health, or whose hæmoglobin value was below the normal range. Yet many donors are advised to resign, because it is thought that blood donation is affecting their health. That advice is, of course, accepted by the transfusion service, but is it justified? Since "anæmia" is a common reason for such advice, it was thought that a full hæmatological investigation of regular donors would give valuable evidence on the effect of blood donation on the hæmatopoietic system.

It is the purpose of this paper to present the results of such an examination of regular donors.

Methods and Materials.

The donors examined comprised 46 males and 32 females. All had made between 45 and 55 donations of blood, each of 400 to 500 millilitres, over periods of eleven to thirteen years. Their ages ranged from thirty to sixty-three years, the average age of the males being forty-eight years and of the females fifty-three years. All the subjects had last given blood approximately twelve weeks prior to collection of samples for investigation. The donors were requested to restrict their previous meal to dry toast and tea or coffee without milk.

Two samples of venous blood were obtained from each subject. The first, for investigation of the cellular elements and the serum proteins, was collected with needle and syringe with light manual constriction of the vein lasting for not longer than thirty seconds. For the blood counts four millilitres of blood were delivered into a tube containing the crystals derived from the evaporation of 0·16 millilitre of a solution containing two grammes of potassium oxalate and three grammes of ammonium oxalate per 100 millilitres. The remainder was allowed to clot, and the serum was used for protein estimations and electrophoresis. The second sample, for serum iron and iron-binding capacity determinations, was collected into an iron-free tube at the end of the blood donation.

Hæmoglobin and hæmatocrit estimations were performed as described by Walsh, Arnold, Lancaster, Coote and Cotter (1953).

Red cell counts were made with two separate 1:200 dilutions of blood in formalin-citrate solution (Dacie, 1950). The dilutions were always made with a single standardized 10 millilitre graduated pipette (for delivering four millilitres of diluting fluid), and with a single calibrated 20 cubic millimetre pipette. A standardized hæmocytometer chamber was employed, and the cells were counted over an area of 0·8 square millimetre (0·4 square millimetre for each dilution).

Blood for the white cell counts was diluted 20 times in a white cell diluting pipette, and the cells were counted over an area of eight square millimetres. Two hundred cells were counted in the differential leucocyte counts.

Reticulocyte preparations were made as described by Dacie, and 2000 cells were counted in every instance.

Serum iron and iron-binding capacity estimations were made by the methods described by Kaldor (1953*a*).

The total serum protein values were determined by the Biuret method of Robinson and Hogden (1940); but readings were made in a photoelectric colorimeter with the use of an "EEL" filter Number 626 with maximum transmission at 570 micromillimetres. Serum protein values were obtained from a graph relating optical density to protein concentration. This graph was constructed from five sera whose protein concentration had been determined by micro-Kjeldahl analysis. The graph was checked towards the end of the survey, but no change had taken place. Paper electrophoresis was performed on all the samples, and the strips were stained for protein (Flynn and de Mayo, 1951). The percentage of the various proteins was determined by scanning a number of the strips in a photoelectric photometer.

Results.

Erythrocytes.

The hæmatological findings are shown in Tables I and II. The mean hæmoglobin and hæmatocrit values are significantly higher than the means reported for New South Wales by Walsh *et alii* (1953). These workers found mean values of 15·76 and 13·93 grammes of hæmoglobin per 100 millilitres, and mean hæmatocrit values of 46·13 and 41·33 *per centum*, for males and females respectively.

The mean values for red cell counts are significantly higher than the values found in many countries as listed by the Advisory Committee on Nutrition Surveys of the Nutrition Society (1945). The weighted means for selected groups in the United States are 5,170,000 per cubic millimetre for 962 male subjects and 4,660,000 per cubic millimetre for 358 female subjects. The only authors to report counts as high as those obtained in the present investigation were Wintrobe and Miller (1929), who found a mean value of 5,870,000 per cubic millimetre for red cell counts on 100 male students (average age twenty to thirty years).

The figures for mean corpuscular hæmoglobin concentration are in good agreement with those found for Australian adults by Lancaster and Cotter (1954) and by McLean (1938). The values for the mean corpuscular volume are considerably lower than those found by Lancaster and Cotter (91·9 cubic microns for males and 91·2 cubic microns for females) and by McLean (89 and 88 cubic microns). Both these groups also found higher values for the mean corpuscular hæmoglobin (Lancaster and Cotter, 31·3 and 30·4 micromicrogrammes; McLean,

[1] This work was undertaken during tenure of a Fellowship of the National Health and Medical Research Council.

TABLE I.

Red Cell and Hæmoglobin Findings in 45 Male and 32 Female Donors.[1]

Subjects.	Red Cell and Hæmoglobin Findings.						
	Erythrocytes. (Millions per Cubic Millimetre.)	Hæmoglobin Value. (Grammes per 100 Millilitres.)	Hæmatocrit Reading. (Percentage.)	Mean Corpuscular Volume. (Cubic Microns.)	Mean Corpuscular Hæmoglobin. (Micromicrogrammes.)	Mean Corpuscular Hæmoglobin Concentration. (Grammes per 100 Millilitres Packed Erythrocytes.)	Reticulocytes. (Percentage.)
Males (45):							
Mean[2]	5·87±0·06 (5·17)	16·31±0·13 (15·76)	47·48±0·38 (46·13)	81·6 ±0·59 (91·9)	27·9 ±0·22 (31·3)	34·5 ±0·15 (34·2)	0·55±0·04
Standard deviation	0·43±0·05	0·90±0·10	2·55±0·27	3·99±0·42	1·47±0·15	1·01±0·11	0·29±0·03
Females (32):							
Mean	5·24±0·07 (4·66)	14·44±0·11 (13·93)	42·79±0·41 (41·33)	82·1 ±0·88 (91·2)	27·7 ±0·29 (30·4)	33·9 ±0·16 (33·4)	0·48±0·04
Standard deviation	0·37±0·05	0·64±0·08	2·29±0·29	4·89±0·62	1·64±0·20	0·92±0·12	0·25±0·03

[1] Throughout this paper, standard errors follow the measure to which they apply.

[2] The mean values shown in parentheses are those of other series as discussed in the text. They are inserted to facilitate comparison.

TABLE II.

Absolute Values for Leucocytes.

Subjects.	Leucocytes: Differential Count per Cubic Millimetre.					
	Total Leucocytes.	Neutrophile Cells.	Lymphocytes.	Monocytes.	Eosinophile Cells.	Basophile Cells.
Males (45):						
Mean	8180±278	4840±214	2560±116	420± 25	210± 38	55± 6
Standard deviation	1867±197	1435±151	778± 82	171± 18	254± 27	43± 5
Females (32):						
Mean	7080±264	4060±209	2380±114	350± 24	170± 21	50± 6
Standard deviation	1496±187	1182±148	646± 81	135± 17	117± 15	35± 4

TABLE III.

Range of Values for Leucocytes.

(Mean±twice standard deviation.)

Subjects.	Leucocytes (per Cubic Millimetre).					
	Total Leucocytes.	Neutrophile Cells.	Lymphocytes.	Monocytes.	Eosinophile Cells.	Basophile Cells.
Males	4400 to 11,900	2000 to 7700	1000 to 4100	80 to 800	0 to 700	0 to 140
Females	4100 to 10,100	1700 to 6400	1100 to 3700	80 to 600	0 to 400	0 to 120

TABLE IV.

Serum Iron Content and Total Iron-Binding Capacity Values.

Observation.	Males.			Females.		
	Number of Subjects.	Mean.	Standard Deviation.	Number of Subjects.	Mean.	Standard Deviation.
Serum iron content (micrograms per 100 millilitres)	46	116±4·7 (124)[1]	32±3·3	29	101±5·2 (108)	28±3·7
Total iron-binding capacity (microgrammes per 100 millilitres)	45	335±8·2 (315)	55±5·8	27	311±9·5 (315)	49±6·7

[1] The mean values shown in parentheses are those of other series as discussed in the text. They are inserted to facilitate comparison.

31 and 29 micromicrogrammes). Furthermore, the values obtained in the present investigation are lower than any listed in the Nutrition Society's review, except when potassium oxalate was used as the anticoagulant.

The mean values for reticulocytes are within the range of 0% to 2%, which is usually regarded as normal.

Leucocytes.

It will be seen from Table II that the total leucocyte count is less for the female subjects than for the males. This is due almost entirely to a higher neutrophile cell count in the males. In general the figures shown are comparable with those found by Blackburn (1947) in 642

male service personnel, and by Osgood, Brownlee, Osgood, Ellis and Cohen (1939) in 269 young adults. The range of values for the leucocytes in the subjects of this survey has been calculated from the mean *plus* and *minus* twice the standard deviation, and is shown in Table III. One male subject was found to have a total eosinophile cell count of 1586 per cubic millimetre. No attempt was made to ascertain the reason for this, but it has obviously increased the mean and the standard deviation of the eosinophile cell count.

Serum Iron Content and Iron-binding Capacity.

Table IV gives the values for serum iron content and total iron-binding capacity. These values are similar to those obtained by Laurell (1947), who found mean serum iron values for normal adults of 124 microgrammes per 100 millilitres in males and 108 microgrammes per 100 millilitres in females, with a total iron-binding capacity of 315 microgrammes per 100 millilitres for both sexes. Kaldor (1953*b*) obtained slightly lower values, but this worker collected blood from the subjects at different times of the day and demonstrated a diurnal variation, the lowest values being obtained in the afternoon. In the present series the mean percentage saturation of the iron-binding protein is 35 for males and 32 for females; the distribution of these values is shown in Figures I and II.

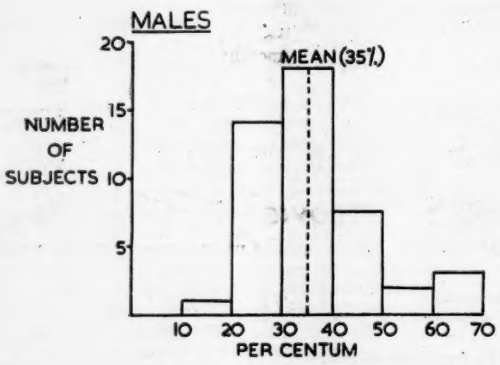

FIGURE I.
Percentage saturation of iron-binding protein: males.

In a series of Swedish blood donors, Laurell obtained a mean serum iron value of 75 microgrammes per 100 millilitres. The total iron-binding capacity was increased, and Laurell concluded that the blood donors were in a state of mild iron deficiency as a result of repeated blood loss. Laurell's donors had given an average of 2500 millilitres of blood in the preceding year, and serum iron values were determined two months after the last donation. These donors apparently did not receive supplementary iron.

One female donor in the present series had a serum iron content of 32 microgrammes per 100 millilitres, a total iron-binding capacity of 360 microgrammes per 100 millilitres, and a percentage saturation value of eight.

Serum Protein.

The results of the serum protein estimations are compared in Table V with those obtained in a series of control subjects. It will be observed that there are no significant differences in either males or females.

Electrophoretic analyses on paper strips were made on all the subjects of the survey. No instance of abnormal distribution of the fractions was found.

Discussion.

The results in this paper demonstrate clearly that the hæmatopoietic system of the blood donors investigated is capable of meeting the extra demands necessitated by the removal of 500 millilitres of blood at regular intervals. During a short period there must be an increased rate of red cell formation and delivery to the circulation, but this probably does not represent more than a 50% increase. It is well known that red cell regeneration may proceed at a much greater rate after pathological hæmorrhage. Coleman, Stevens, Dodge and Finch (1953) have shown that if sufficient iron is supplied, the hæmatopoietic system of normal subjects is capable of maintaining relatively normal hæmatocrit values after removal of 500 millilitres of blood at fortnightly intervals for eight months. These workers demonstrated clearly that the factor limiting the

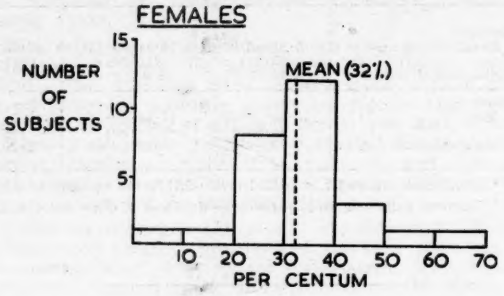

FIGURE II.
Percentage saturation of iron-binding protein: females.

recovery of the hæmatocrit value after hæmorrhage is usually the amount of available iron and not the hæmatopoietic tissue. It is the practice of the New South Wales Red Cross Blood Transfusion Service to supply all blood donors with 15 tablets containing 0·2 gramme of ferrous sulphate. They are advised to ingest one tablet daily after giving blood. Laurell (1947) did find some evidence of iron deficiency in Swedish blood donors, but as has been

TABLE V.
Serum Protein Values.

Subjects.	Serum Protein Values. (Grammes per 100 Millilitres.)	
	Mean.	Standard Deviation.
Males:		
Controls (18)	6·49±0·11	0·47±0·08
Blood donors (46)	6·68±0·06	0·43±0·04
Females:		
Controls (24)	6·37±0·09	0·45±0·06
Blood donors (32)	6·56±0·05	0·30±0·04

pointed out already, no supplementary iron was given to these donors. It is also of interest to note that in the present series the one female donor with a low serum iron value did not take the prescribed iron tablets. Iron is certainly prescribed too freely in medical practice. It is not generally appreciated that iron is excreted from the body only in minute amounts, and that this small loss is usually replaced from the diet without difficulty. However, hæmorrhage or blood donation results in the loss of a considerable amount of iron, and it is reasonable to supplement the dietary iron by giving tablets of ferrous sulphate.

There are two interesting features of the hæmatological results. The first is the significant increase in the mean values for red cells, hæmoglobin and hæmatocrit, the value for the red cells being proportionately higher than the values for hæmoglobin and hæmatocrit. The second observation is the finding of lower values for the mean corpuscular volume and the mean corpuscular hæmoglobin and is dependent on the relatively higher red cell count.

It is difficult to explain these findings. After recovery from the loss of 500 millilitres of blood, a change takes place in the red cell population. Red cells of mixed ages,

varying from one to 120 days, are removed in the 500 millilitres of blood, and are replaced within a short time by an equivalent number of new red cells, each with an expected life span of 120 days. This means that the average age of the red cell population in the subject will be less than normal, and that a smaller percentage of the red cells will each day reach the end of their normal life span. The rate of red cell destruction per day will therefore be reduced. If hæmatopoiesis proceeds at the usual rate for the subject and is not influenced by this relatively minor reduction in the number of red cells destroyed, it is not unreasonable to suppose that there will be a small increase in the absolute number of red cells ninety days after removal of blood. This may account for the high values observed in the present series. It must, however, be admitted that no evidence has been produced to substantiate this hypothesis.

It should also be mentioned that blood samples were obtained from all subjects during the winter months. It has been shown that hæmoglobin values of comparable groups in relation to age and social factors are higher in the winter than in summer in New South Wales (Walsh *et alii*, 1953).

No satisfactory explanation can be offered for the low values obtained for the mean corpuscular volume and the mean corpuscular hæmoglobin.

It must be admitted that the donors investigated in this survey may not be representative of all the volunteers who enrol as donors. The possibility cannot be overlooked that those volunteers who discontinue are unable to maintain normal hæmatological values, and that those people who continue are able to do so. However, investigations have shown that donors discontinue for a variety of reasons, and that ill-health is not more frequent among those who default after the tenth or twentieth donation than after the first or second. It seems probable that sustained interest and activity in blood donors are more a matter of psychology than of physiology.

Summary.

The effect of regular removal of blood has been investigated in 46 male and 32 female blood donors, who had all given more than 20 litres of blood. The mean hæmoglobin, hæmatocrit and red cell values were higher than the mean values of the general population in New South Wales, whilst the mean corpuscular volume and mean corpuscular hæmoglobin values were lower. Values for the serum iron and iron-binding capacity measurements were within the accepted range of normality, indicating the absence of iron deficiency in the donors. No abnormality was detected in the serum proteins. It is apparent that regular blood donations have had no harmful effect on the health of these donors.

Acknowledgements.

The writer wishes to acknowledge the technical assistance of Mrs. Pamela Price and Miss Marjorie Powell.

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DEATHS FROM MALARIA ON THE AUSTRALIAN MAINLAND.

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THE number of deaths from malaria in Australia in the seven years 1946 to 1952, following the second World War, amounted to more than half of the number of deaths due to this disease in Australian Army patients during that war.

Table I shows the figures for the total number of deaths from malaria in Australia in the years 1908 to 1952. It will be seen that deaths from this disease are steadily declining. However, this satisfactory decrease in the total number of deaths should not make us so complacent as to ignore the comparison in case fatality rates shown in Table II. It will be seen that the figures for Europeans in Papua-New Guinea are ten times as large as the wartime army figures. This rate for Papua-New Guinea is derived from cases of malaria in administration hospitals. In Australia (except New South Wales, where malaria was not notifiable during this period), the case fatality rate of 8·9 per 1000 is 23 times as large as the wartime army figures, and in 1952 there were nine deaths from malaria and approximately 41 reported cases.

TABLE I.

Deaths Due to Malaria in All Australian States and the Northern Territory for the Years 1908 to 1952.

Years.	Deaths.			Average Deaths per Annum.
	Males.	Females.	Total.	
1908 to 1910 ..	141	25	166	55·3
1911 to 1920 ..	231	74	305	30·5
1921 to 1930 ..	209	49	258	25·8
1931 to 1940 ..	125	24	149	14·9
1941 to 1945 ..	48	10	58	11·6
1946 to 1952 ..	34	13	47	7·8

TABLE II.

Comparison of Case Fatality Rates in the Australian Army, Amongst European Patients in Papua-New Guinea, and in Australia.

Group.	Years.	Case Fatality Rate per Thousand.
Australian Army	1939 to 1945	0·38
Europeans in Papua-New Guinea ..	1947 to 1952	3·6
Australia (except New South Wales)[1] ..	1947 to 1952	8·9

[1] Malaria was not notifiable in New South Wales.

New South Wales, in the period from 1947 to 1952, contributed one-third of the total number of deaths due to malaria in the whole of Australia; but the number of cases is unknown. This number would be expected to be considerably less than in Queensland and the Northern

Territory, so that the case fatality rate in this State is probably higher than the figure given for the rest of Australia. In this regard it is regrettable that malaria is not a notifiable disease in New South Wales.

This increase in the case fatality rate requires study, as the figure quoted above for 1952 is alarming; it would almost indicate that malignant tertian malaria is a fatal disease in Australia. In Papua-New Guinea there are many non-fatal attacks of malaria which are treated by the patient himself, so that the figure given for that area for administration hospital patients is for a selection of the more serious cases.

It should be pointed out, in the first place, that benign tertian malaria in the south-west Pacific area is rarely a cause of death in a person who has been in moderately good health. Malignant tertian is the type of malaria which kills. The excellent results of treatment in the wartime Australian Army were due to several factors, probably the most important of which was the fact that medical officers and, indeed, the troops themselves were alert to the presence and danger of the disease. This resulted in early diagnosis and treatment. Partial suppression may also have contributed.

Malignant tertian malaria, which is probably always an imported disease in Australia, will not be diagnosed and therefore will be fatal unless it is always kept in mind. In 1947 and 1948 there were approximately 2500 cases of malaria reported in Australia (excluding New South Wales), with 12 deaths. In the following four years there were approximately 250 cases reported with 13 deaths—a fatality rate of 52 per 1000—that is, 140 times the wartime army figures. This demonstrates the declining awareness of the disease, for, unlike the resistance to antibiotics which has appeared with some organisms, the malaria parasites have not acquired resistance to the antimalarial drugs used for treatment.

A considerable number of medical practitioners saw sufficient cases of malaria during war service to become aware of the protean manifestations of the disease; but this experience has faded into the background, and as malaria is not endemic in the southern parts of Australia, it is not usually considered in diagnosis. Medical students at the University of Sydney receive instruction on the subject; but there is a large body of younger graduates who have not had the wide experience of malaria gained by service medical officers in the war years. A parallel may be drawn with typhoid fever, which was formerly a common disease in Australia, but now has become rare. However, typhoid became well entrenched in the thought pattern of differential diagnosis, and is well remembered today. Malaria might well take a place beside typhoid in the list of diseases which will not be diagnosed if they are not considered.

Experience with cases of locally acquired benign tertian malaria indicates the delay which may occur between the onset of symptoms and diagnosis. In a case reported by Collier (1944) the patient had been ill for sixteen days, and in another reported by Morgan (1934) for over five weeks before a diagnosis was made. Patients suffering from benign tertian malaria can survive this time; but with malignant tertian infections of New Guinea origin it was estimated by Fairley and his colleagues (1947) that death would take place on the twenty-second day after infection—that is, twelve days from the first onset of symptoms—if no treatment was given.

The European population of Papua-New Guinea is considerably larger than in pre-war years. Aerial travel has taken the place of travel by sea. There are consequently now many more people in Australia than in pre-war years who have resided in these highly malarious areas and have arrived well within the incubation period of malignant tertian malaria. There are, too, people on leave from Netherlands New Guinea, the British Solomon Islands and the New Hebrides—all highly malarious countries. Another factor which distinguishes the post-war years is the effectiveness of malarial suppression. A certain proportion of residents in malarious areas in post-war years have never had an attack of malaria. In Australia they may not recognize the symptoms of onset when an attack occurs, and are thus unable to call the medical officer's attention to the possibility of malaria as the diagnosis of their illness.

There are others besides island residents on leave who may present themselves suffering from malaria. Numerous tourists, scientific workers, business executives, personnel of moving picture companies, government officials, airline employees, sportsmen, politicians and seamen visit the south-west Pacific islands and other malarious countries and must be regarded as having been exposed to the risk of infection. These are the potential patients who will die from malignant tertian malaria unless the possibility of its occurrence is remembered.

Malaria transmitted on the Australian mainland during recent years has mainly been of the benign tertian variety; but there have been invasions by the malignant tertian parasite in the past. About twenty years ago over 200 aborigines and white people died during a malignant tertian epidemic in the north of Western Australia before the correct diagnosis was established.

During the war it was not until the Commander-in-Chief was made aware of the importance of malaria that control of this disease in the Australian Army became effective. In peacetime conditions in malarious countries it is only when the administrations become aware of the health and economic importance of the disease that any worthwhile malaria control measures are taken. In Australia today a small number of people will probably continue to die from malignant tertian malaria if the medical profession does not acquire an awareness of the disease. As a supplementary measure, it may be possible for a suitable pamphlet to be prepared on the subject of malaria and the possibility of its first appearing on return to Australia. This could be handed to people entering from malarious areas by the customs officer at the port or airport of entry. A more general pamphlet was at one time prepared for all persons intending to enter Papua-New Guinea from Australia.

Acknowledgement.

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ACCIDENTAL INJURIES IN PRE-SCHOOL CHILDREN: II. TRAFFIC ACCIDENTS.

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THE first article in this series (Clements, 1955) gave a general survey of the accidents sustained from birth to the sixth birthday by a group of 12,131 boys and 11,653 girls who were the subjects of a special study. Details of the methods used to collect and analyse the data are given in that article.

An analysis of the accidents due to vehicular traffic which the children in this study sustained is reported here.

[1] Endowed by the Commonwealth Department of Health.

Results.

We have followed the official statistics in separating the traffic accidents into two classes, according to whether the child had been a passenger in or on a vehicle or had been a pedestrian or cyclist. Table I shows the numbers of each type of accident for each year of age sustained by children as passengers. Although seven categories of accident are listed in this table, it may be noted that in only three of these is the child apparently an innocent party—that is, when a passenger in a motor-car or on a cycle when that vehicle is involved in an accident. These three types of accident accounted for about 48% for boys and 40% for girls of the accidents in this group. In the other four categories the child had some part in producing the accident. Most, if not all, the latter accidents were easily preventible.

Table II shows the numbers of accidents sustained by children when pedestrians or cyclists. The pedestrian group is divided into two subgroups; this for some accidents was an arbitrary decision. The details of the story as written by the parent decided the subgroup into which the particular accident was placed. To some extent a mother's sense of guilt might have biased her attitude. Few accidents occurred to cyclists, largely because of the age of the group.

None of the accidents recorded in this study was fatal. However, an attempt was made to separate the more serious from the mild accidents. The children who sustained a fractured skull or fractured limb or cerebral concussion with unconsciousness, or severe lacerations, were placed in the former group. The remainder were considered to have suffered mild effects. The numbers for the two types of accident are given in Tables III and IV. In the preparation of Table III only the categories with a significant number of children involved have been included. It may be noted that when the child was a passenger, the serious type of accident accounted for 14% and 16% for boys and girls respectively of all accidents in the series, whereas this type of accident was responsible for 27% and 19% respectively of the accidents to boys and girls who were pedestrians or cyclists.

For comparison of certain aspects of these data, a table has been prepared from information given in the "Annual Reports of the Superintendent of Motor Transport, N.S.W. Department of Road Transport", for the years 1950 to 1953. These reports give the numbers of children injured and killed in traffic accidents in New South Wales, and also indicate whether they were passengers, pedestrians or cyclists. Table V compares these data with those obtained in this survey. The official statistics are cumulative over four years, some of which coincided with this period of this survey. A comparison of the two sets of figures is of little value; however, the things which are of interest are the ratios of passengers to pedestrians, of the percentage of killed to total injured in the official statistics, and of the percentage of severely injured to total injured in this study. These ratios and percentages are also shown in this table.

Discussion.

In the last decade traffic accidents have been the second most important cause of accidental deaths in pre-school children, being surpassed by drowning. In earlier periods in this century burns and scalds, and drowning, were both responsible for more deaths in this age group. In the one to four years age group deaths from burns and scalds have been greatly reduced over the last thirty years, but death rates from traffic accidents have increased; whereas the death rates for traffic accidents for the children aged five to nine years have remained fairly stationary, and those for children aged ten to fourteen years have shown a considerable fall. This difference in the trends of rates of the last forty years is almost certainly a reflection of the road safety campaigns conducted through the schools (Clements, 1952).

In this survey, traffic accidents occupy fourth place in order of magnitude, being surpassed by falls, burns and scalds and accidental poisoning. However, traffic accidents to children do constitute an important group, for whereas the circumstances surrounding many other types of accidents are peculiar to childhood (for example, burns and accidental poisoning), involvement with road traffic in some way is a constituent part of modern civilization. Measures designed to prevent road traffic accidents to children, if properly laid, should have lasting effects upon the total problem of traffic accidents.

The situation of the child in relation to the accident is important for the development of preventive measures. It will be noted from Table V that relatively more children in this survey were involved as passengers than in the accidents reported by the Department of Road Transport. This could be explained by the large number of accidents of a relatively minor character recorded in this survey in which only the child was involved; most of these accidents would not be reported to the authorities. This group includes those accidents which resulted from the child's opening of the car door, and from the child's falling through a window or having its hand or limb caught in a door. If this group is excluded from the totals, the ratio of children as passengers to children as pedestrians in this

TABLE I.
Vehicular Accidents Including Children as Passengers.

Type of Accident.	Sex.	Number of Accidents at Each Year of Age.						Total Number of Accidents.
		Under One Year.	One Year.	Two Years.	Three Years.	Four Years.	Five Years.	
Car in collision with another vehicle ..	M.	9	7	27	39	40	20	142
	F.	1	10	16	25	19	14	85
Child opened door and fell out	M.	1	9	27	31	24	13	105
	F.	1	5	23	19	18	14	80
Child fell through open window	M.	1	2	—	1	—	—	4
	F.	—	1	—	—	—	—	1
Child's fingers or limbs jambed in door ..	M.	—	1	9	21	12	4	47
	F.	3	4	5	18	12	4	46
Child released brakes and car damaged, child injured.	M.	—	1	—	1	—	—	2
	F.	—	1	1	—	—	1	3
Child as passenger on motor-cycle	M.	1	—	—	1	1	—	3
	F.	1	—	2	—	—	—	3
Child as passenger on push-bicycle ..	M.	—	—	1	—	2	1	4
	F.	—	—	—	—	—	—	—
Totals	M.	12	20	64	94	79	38	307
	F.	6	21	47	62	49	33	218
Number of children exposed at each year of age	M.	12,131	11,727	10,690	10,690	10,690	10,690	—
	F.	11,653	11,481	10,583	10,583	10,583	10,583	—

TABLE II.

Vehicular Accidents to Children when Pedestrians or Cyclists.

Type of Accident.	Sex.	Total Number.	Number of Accidents at Each Year of Age.					
			Under One Year.	One Year.	Two Years.	Three Years.	Four Years.	Five Years.
Child running heedlessly on road—hit by car, motor-cycle or push-bicycle	M.	46	—	5	2	9	19	11
	F.	28	—	2	3	7	10	6
Child playing on road—hit by car, motor-cycle or push-bicycle.	M.	127	1	2	17	27	41	40
	F.	86	5	2	6	27	24	22
Child riding push-bicycle or scooter on road, hit by car, motor-cycle or push-bicycle.	M.	16	—	—	—	4	3	9
	F.	5	—	—	1	2	2	—

TABLE III.

Severity of Vehicular Accidents Involving Children as Passengers.

Type of Accident.	Sex.	Total Number of Accidents.	Number of Mild Accidents.	Number of Serious Accidents.	Number of Each Type of Serious Accident.			
					Fractured Skull.	Fractured Limb.	Concussion.	Severe Lacerations.
Car in collision with another vehicle ..	M.	142	121	21	5	7	4	5
	F.	85	67	18	2	7	1	8
Child opened door and fell out	M.	105	91	14	2	4	7	1
	F.	80	70	10	4	1	3	2
Child's finger or limb jambed in door ..	M.	47	41	6	—	3	—	3
	F.	46	40	6	—	6	—	—
Totals	M.	294	253	41	7	14	11	9
	F.	211	177	34	6	14	4	10

TABLE IV.

Severity of Vehicular Accidents to Children when Pedestrians or Cyclists.

Type of Accident.	Sex.	Total Number of Accidents.	Number of Mild Accidents.	Number of Serious Accidents.	Numbers of Each Type of Serious Accident.			
					Fractured Skull.	Fractured Limb.	Concussion.	Severe Lacerations.
Child running heedlessly on the road—hit by car, motor-cycle or push-bicycle.	M.	46	30	16	3	12	—	1
	F.	28	24	4	—	—	2	2
Child playing on road—hit by car, motor-cycle or push-bicycle.	M.	128	95	33	8	18	4	3
	F.	86	67	19	5	14	—	—
Child riding push-bicycle or scooter on road—hit by car, motor-cycle or push-bicycle.	M.	16	14	2	—	2	—	—
	F.	5	5	—	—	—	—	—
Totals	M.	190	139	51	11	32	4	4
	F.	119	96	23	5	14	2	2

survey (namely, boys 1:1·1, girls 1:1·2) approaches much more nearly the ratios in the official statistics.

This survey has thus revealed an important type of accidents which are not normally reported to the authorities; as was pointed out above, these amount to approximately half the accidents in this group. Some 12% of the children involved in these accidents were severely injured; this is of the same order as the percentage severely injured when the motor vehicle was involved in an accident.

Table I shows a strong masculinity in the accidents with significant numbers, except those in which the child has its hand jambed in a door. The masculinity of traffic accidents has been noted in other surveys, and it has been assumed that it is the result of the natural attitude of boys, even at this age, to explore and get into mischief. The high masculinity in accidents in which the vehicle is involved is hard to explain, unless we accept the suggestion that there are more male drivers on the road at any one time, and that whereas mothers take their children in the car with them, fathers tend to take their sons in preference to their daughters. It seems that boys and girls suffer equally from carelessness in the closing of doors.

After the second birthday, when the child has become mobile and can sit in the car unsupported, the rates for all accidents involving the child as a passenger rise, with sharp peaks at the ages of three and four years, followed by a decline in the fifth year, when the child has apparently learned some of the hazards of the car.

The accidents sustained by children running on to or playing on the road show a progressive rise from two to five years of age. As was pointed out above, it is not easy to draw a clear distinction between these two types of accident, and perhaps the most satisfactory procedure is to consider them together. The ratio of boys to girls at each age is almost 2:1. This difference cannot be explained on the basis of different methods of play, for authorities on child development maintain that in this age group the play activities of boys and girls are similar. A possible explanation is to be found in the associations

TABLE V.

Comparison of Road Accidents from Official Statistics and This Survey.

Situation of Child in Relation to the Accident.	From the Annual Reports on Road Accidents in New South Wales for Years 1950 to 1953.						This Survey.					
	Male.			Female.			Male.			Female.		
	Injured.	Killed.	Killed. (Percentage of Total Injured.)	Injured.	Killed.	Killed. (Percentage of Total Injured.)	Total Injured.	Severely Injured.	Severely Injured. (Percentage of Total Injured.)	Total Injured.	Severely Injured.	Severely Injured. (Percentage of Total Injured.)
As passenger	471	25	5·0	320	10	3·0	300	41	14	215	34	16
As pedestrian	570	41	6·7	296	23	7·2	173	51	29	114	23	20
As cyclist ..	5	—	—	—	—	—	16	—	—	5	—	—
Ratio, passengers to pedestrians	1:1·2	1:1·6	—	1:0·9	1:2·3	—	1:0·57	1:1·2	—	1:0·53	1:0·75	—

of the two sexes. In general, small boys tend to play with their older brothers or older boys from the neighbourhood who make the street a playground; young girls tend to seek their mother's company or that of older girls who do not usually play in the street.

From Table V it will be seen that there is a higher percentage of deaths in the official statistics and a higher percentage of severely injured children in this survey, amongst children injured as pedestrians, compared with those injured as passengers.

Prevention of Traffic Accidents.

Dietrich (1950), who has studied techniques for the prevention of accidents in childhood, has expanded the concept that parental action in this area has two components, protection and education. During the first year of life, the only action is protection; however, this should be progressively withdrawn as the child is taught through the educational efforts of its mother to take care of itself. The timing of this withdrawal will obviously vary with both the age of the child and the situation. Education is used here in the broad sense to define the process by which the child develops independence of thought and action. The accidents discussed in this survey occurred before the children went to school; it is noteworthy that death rates for traffic accidents are higher in the one to four years age group than in either the five to nine or ten to fourteen years age groups. Education directed towards accident prevention in this age group is the responsibility of parents. Traffic accidents can be divided into three categories for the development of procedures which aim at preventing their occurrence.

The Child as a Passenger in a Car Involved in an Accident.—When a child is a passenger in a car involved in an accident, seldom is the child more than the innocent victim. Prevention of this type of accident is bound up with the over-all improvement in road safety. It is probable that some of these accidents arise from the driver's distraction by an unruly child. It would be interesting to know how often this occurs. This information was not available in this survey, but it might be obtained by the authorities in the inquiries into traffic accidents.

Accidents Due to the Child's Opening a Door or Falling from a Window.—It has already been pointed out that the peak of accidents due to a child's opening a car door or falling from a window is in the three and four years age group; most five-year-olds have learnt of this hazard. Some degree of protection, in the form of deliberate action by the driver to fasten the safety catch, must be maintained, when any pre-school child is travelling in a car; however, it would seem that this is particularly necessary with three and four year old children. Education about the hazards of a door should be commenced in the third year. Every car journey should be used as a teaching and learning experience. Words alone are not enough; demonstrations should be made by a passenger of what happens when somebody plays with the door fastenings while the car is in motion. These are of more value than a series of "don'ts". Similar dual action should be taken to prevent children from leaning out of the car window while the car is in motion. For young children the windows should be closed as a protective measure; for older children advice and demonstration of how easily a doll or some similar article can fall from a window should form the basis of the educational component.

Accidents that Occur to Children as Pedestrians.—Young children must be protected by constant adult attention and supervision; but education should be started in the toddler stage. The mother should use each walk on the street with the child as a teaching experience. She should fully obey traffic regulations, explaining to the child why she is walking on the footpath, why she crosses the road in the approved manner, *et cetera*. After a few lessons the child can be asked to indicate where they should walk, when and how they should cross the road. These simple procedures should develop in the child a strong road sense long before he goes to school.

Summary.

1. The traffic accidents which occurred among 12,131 boys and 11,653 girls, aged under six years, who were the subjects of a special accident survey, are reported here. These types of accidents occupy fourth place in magnitude in this study, being exceeded by falls, burns and scalds and accidental poisonings.

2. Of these totals, 307 (2·4%) boys and 218 (1·9%) girls were involved in a vehicular accident as passengers; 189 (1·4%) boys and 119 (1·0%) girls suffered as pedestrians or cyclists.

3. Of the children involved in vehicular accidents as passengers, 14% of boys and 16% of girls were seriously injured, and of those involved as pedestrians or cyclists 26% of boys and 20% of girls were seriously injured.

4. The prevention of these types of accidents is discussed.

Acknowledgements.

I have to thank Dr. H. O. Lancaster for statistical advice; Mrs. Goldstein, Miss M. Reid, Miss N. Holloway and Miss M. George for valuable assistance in the sorting and analysis of the cards; and the many kindergarten directors and school teachers whose enthusiasm and application made this survey possible.

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Reports of Cases.

HEPATIC NECROSIS IN NEONATAL HERPES SIMPLEX INFECTION.

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FATAL hepatic necrosis can be caused by *herpes simplex* virus, as was suggested by Hass in 1935. He recorded the clinical notes and autopsy findings of a premature baby, who died aged fourteen days. Areas of necrosis were present in liver and adrenal glands, and histological examination revealed, in the tissue surrounding these areas of necrosis, the presence of nuclear inclusions identical with those described in *herpes simplex* lesions. The literature presents a total of nine babies who died in the neonatal period from acute hepatic necrosis, the histological appearance of which has suggested an infection with *herpes simplex* virus. This virus has been isolated from liver tissue on two occasions.

We wish to record the case histories and autopsy reports of two babies, each of whom died with severe hepatic necrosis. Nuclear inclusions were present in liver cells in both, and from the liver of one child (B., Case II) a virus identical with that of *herpes simplex* was recovered.

Case I.

A. was admitted to the Royal Children's Hospital when aged twenty hours. The mother's pregnancy, which was her first, had been complicated by severe albuminuria, and was terminated six weeks prematurely by Cæsarean section. His birth weight was three pounds ten ounces. Respirations commenced immediately after delivery, but several cyanotic episodes occurred prior to his transfer to the Royal Children's Hospital. Examination revealed him to be slightly cyanosed, but with the administration of oxygen his colour appeared satisfactory, although the respirations were rapid and the respiratory excursion was poor. His liver was just palpable, but his spleen could not be felt. He was considered to have pulmonary atelectasis, and apart from the continuous administration of oxygen, penicillin and vitamin *K* were given. On the second day after his admission to the hospital feedings of expressed breast milk were commenced. These feedings were taken well. Jaundice, which was just evident on his admission to hospital, became more obvious at this stage. Six days after admission, when his general condition was considerably improved, a generalized macular erythematous rash became obvious. This faded rapidly and disappeared within twenty-four hours. Eight days after his admission he appeared cyanosed, slightly icteric and lethargic. His respirations were slow, and no further changes occurred in his condition until he suddenly died when aged nine days.

Autopsy Findings.

Autopsy was performed twenty-seven hours after death. At this time no skin lesions were visible. The central nervous system appeared normal, as did the heart and great vessels. The lungs were poorly expanded and were of uniform dark red colour apart from pink air-containing areas along their anterior margins. The alimentary tract appeared normal from œsophagus to rectum. The liver was of normal size, but beneath its capsule numerous minute pale areas could be seen. When sections of the organ were prepared, these pale areas were seen to be present throughout its substance (Figure I). Their size varied, but they were all less than two millimetres in diameter. No other abnormalities were detected in the liver, the external surface of which was quite smooth.

The gall-bladder, extrahepatic biliary system and pancreas were normal in appearance. The spleen was slightly enlarged, but no abnormalities were seen in sections prepared from it. The adrenal, pituitary and thyroid glands appeared normal, as did the kidneys, ureters, bladder and genitalia.

Histological Findings.—The myocardium, pancreas and kidneys were normal. The lungs were congested, and alveolar collapse was present. Examination of sections of liver revealed numerous widespread areas of liver-cell necrosis (Figure II). These did not appear to have any zonal distribution. An occasional binucleated liver cell was present, and the portal tracts were infiltrated with mononuclear cells. In the nuclei of the majority of liver cells surrounding the areas of necrosis inclusion bodies could be seen (Figures III and IV). The inclusions varied in appearance from large single eosinophilic masses with margination of nuclear chromatin and the formation of a "halo" to minute pink granules distributed throughout a vesicular nucleus. Other nuclei appeared to be filled with an inclusion of ground-glass appearance, which had a slightly basophilic tinge. The cytoplasm of cells whose nuclei contained inclusions usually presented a degenerate appearance. Similar small areas of necrosis were present in the adrenal cortex (Figure V), and many of the nuclei of the adjacent cells contained inclusion bodies.

No virus studies were made as the possibility that the ætiological agent might be *herpes simplex* virus was realized only after histological preparations had been examined.

Case II.

B., a female child, was born as a result of the mother's first pregnancy. Pregnancy and labour were normal, apart from an attack of "influenza" eight to nine days before term, when the mother suffered from headache, sore throat, lassitude and fever for two days, but recovered without treatment. Labour lasted fifteen hours; the baby presented by the vertex and weighed seven pounds. The baby was well until the sixth day, when she developed a temperature of 102° F., vomiting and slight chest retraction. The provisional diagnosis was atelectasis and dehydration, for which antibiotics were administered. No improvement resulted, and a sudden deterioration in her condition on the ninth day of life was accompanied by pallor and an extremely rapid pulse rate. Her liver was now readily palpable, having been impalpable the previous day. She was transferred to the Royal Children's Hospital with a provisional diagnosis of subcapsular hæmatoma of the liver.

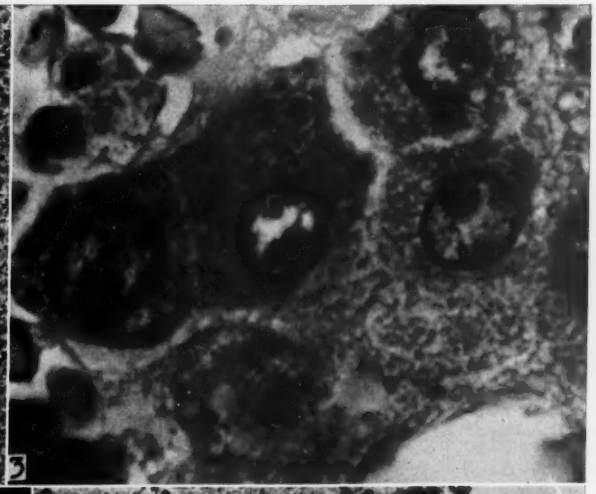
On her admission to hospital, the hæmoglobin value of her blood was 60% (8·7 grammes per 100 millilitres), and a transfusion of blood—O (IV), Rh-positive—was administered. Investigation of the peripheral blood after transfusion revealed the following findings: the hæmoglobulin value was 120% (17·4 grammes per 100 millilitres); the leucocytes numbered 14,400 per cubic millimetre, 51% being neutrophile polymorphonuclear cells, 1% eosinophile polymorphonuclear cells, 1% monocytes and 47% lymphocytes; the platelet count was less than 50,000 per cubic millimetre. The pulse rate became slow, the temperature failed to register and bright blood was passed rectally. She was considered by Dr. John H. Colebatch, who was physician in charge of the case, to be suffering from an acute generalized infection, possibly viral, contracted from the mother before or after birth, and affecting the respiratory system, liver and bone marrow with resulting thrombocytopenia and intraabdominal hæmorrhage. Treatment included the administration of ACTH (20 units *statim*, 10 units every six hours) and the intravenous administration of "Aureomycin" (20 milligrammes every three hours). Death occurred nine hours after her admission to hospital, at the age of nine days.

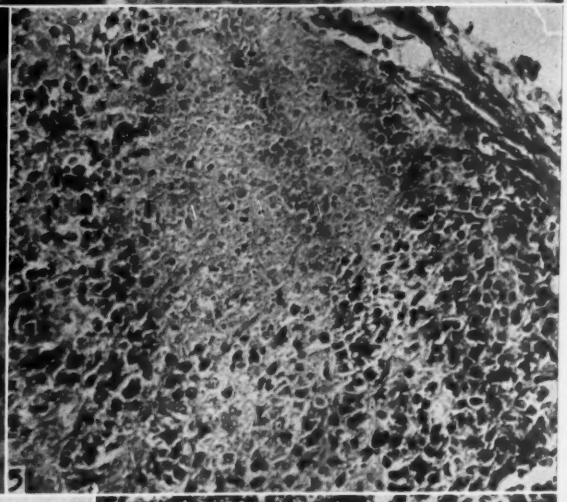
Autopsy Findings.

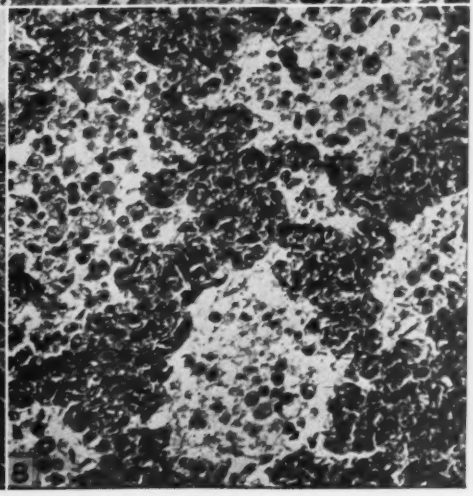
Autopsy was performed twelve hours after death.

The liver (210 grammes) was considerably enlarged and of deep plum colour. Beneath the capsule, which was smooth, numerous small pale areas were visible. Their presence throughout the liver was confirmed when sections were prepared from it. Petechial hæmorrhages were present

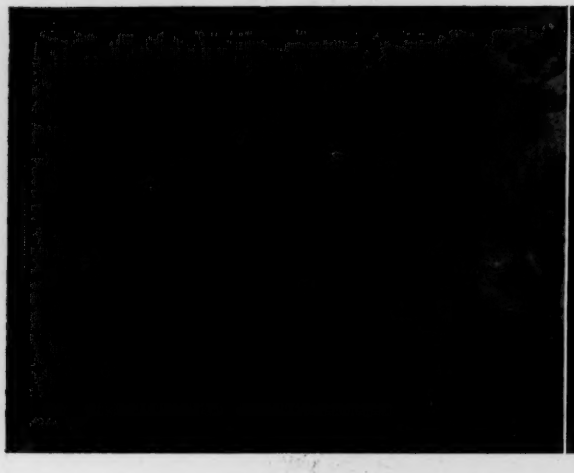
ILLUSTRATIONS TO THE ARTICLE BY ALAN WILLIAMS, M.B., B.S., AND IAN JACK, M.Sc.







ILLUSTRATIONS TO THE ARTICLE BY N. D. HICKS, M.B., B.S., AND O. W. LEITCH, M.S., F.R.A.C.S.



in skin and serous membranes, and all serous cavities contained an increased amount of fluid, the peritoneal cavity containing approximately 100 millilitres of blood-stained serous fluid. Apart from superficial ulceration of the œsophageal mucosa, no abnormalities were present in the alimentary tract. Areas suggestive of hæmorrhage were present in the lung parenchyma. The remainder of the viscera were normal on macroscopic examination.

Histological Findings.—Histological examination of sections revealed the liver architecture to be disorganized by areas of necrosis and hæmorrhage. The reason for the hæmorrhage in the substance of the liver which caused its enlargement was apparent on examination of sections, when necrosis of walls of blood vessels was obvious (Figure VI). No tissue reaction to the necrosis was present, but in the liver cells surrounding these areas numerous intranuclear inclusions were visible. Similar inclusions were present in the cells surrounding an area of necrosis in the adrenal cortex, in the œsophageal mucosa, the cells of which appeared swollen and vacuolated and in some areas desquamated (Figure VII), and in mononuclear cells present in lung alveoli (Figure VIII). A portion of marrow was examined, but no necrosis was apparent, and inclusion bodies were not detected in sections of the heart, trachea, small intestine, spleen, brain, kidney, pancreas or pituitary.

Isolation of Virus.—A small piece (about one gramme) of liver was ground with sterile alundum and a 10% suspension prepared in ice-cold 0·5% gelatine saline containing M/20 phosphate buffer (pH 7·2). The supernate from the lightly centrifuged suspension was inoculated, together with penicillin and streptomycin in 0·05 millilitre amounts, to a litter of eight thirty-six hours old mice. By the third day the resulting infection had caused the death of most of the animals. Two moribund survivors were used to prepare a 10% suspension of eviscerated carcass tissue. By the use of this as an inoculum, the agent was successfully transmitted by sub-passage to suckling mice, to weanling mice, and to the cornea of a rabbit, with the production of lesions characteristic of *herpes simplex* infections. The kerato-conjunctivitis and meningo-encephalitis were confirmed histologically. Typical pocks on the chorio-allantois of eleven-day chick embryos were produced by the original liver inoculum. The virus was further identified as a strain of *herpes simplex* by complement fixation, with the use of an antigen prepared from infected chorio-allantoic membranes of eleven-day chick embryos and from known "positive" human and animal sera, with "negative" controls.

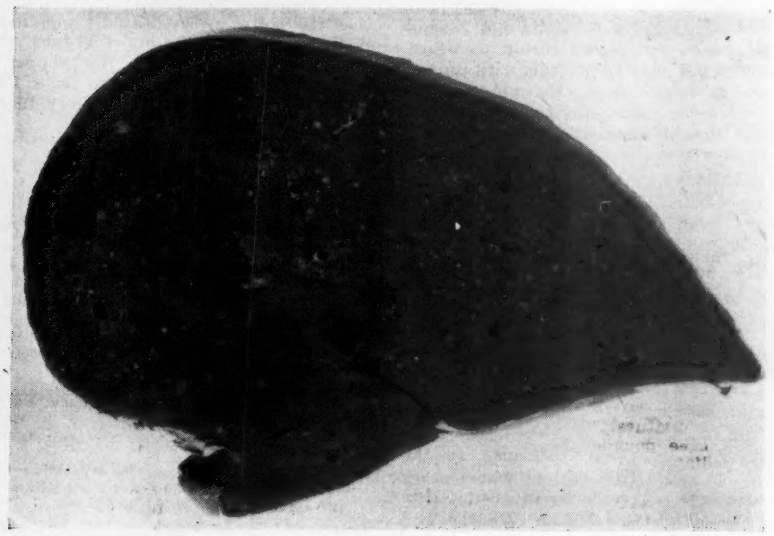


FIGURE I.
Photograph of liver from Case I, showing numerous irregular areas of necrosis in the cut surface.

Comment.

Primary infection with *herpes simplex* virus is common in children, the usual lesion being stomatitis which resolves after two or three weeks. Whilst it is rare, because of immunity transferred from the mother, for infection with this virus to occur before the age of eight months (Anderson and Hamilton, 1949), its occasional occurrence has been proven. In 1941, Smith, Lennette and Reames isolated *herpes simplex* virus from the brain of a baby who died from encephalitis when aged five weeks. Wildi (1951) records the case of a full-term infant who died at the age of eleven days with encephalitis due to *herpes simplex*, the mother having had genital herpes during the fifth month of pregnancy. Florman and Mindlin (1951) describe the isolation of *herpes simplex* virus from the skin lesions of a premature baby, one of triplets, who developed vesicular stomatitis on the eleventh day of life, and subsequently a generalized vesicular exanthem, encephalitis and chorio-retinitis. One other of these triplets had chorio-retinitis, but the third had no obvious lesions.

In 1951 Quilligan and Wilson recorded the case of a baby, aged twelve days, with herpetic lesions of skin and hepatic necrosis. Nuclear inclusions were present in liver cells, and *herpes simplex* virus was isolated from both liver and skin. The mother of this baby had *herpes labialis* nine days prior to delivery.

Zuelzer and Stulberg (1952) presented eight infants with visceral lesions due to *herpes simplex*. They divided these patients into two groups. (*a*) This group comprised five babies, four of whom were born prematurely, who died between the sixth and thirteenth days of life. Apparently normal for the first few days of life, they became icteric, drowsy, febrile and dyspnœic and developed circulatory failure. This state was associated in all cases with hæmorrhage from puncture sites, or from the alimentary canal. Hepatic enlargement was mentioned in two cases only. Three of these babies had herpetic conjunctivitis. At post-mortem examination areas of necrosis were obvious in the liver, and histological studies revealed similar areas in other viscera. These viscera included adrenal, kidney, spleen, lungs, bone marrow, œsophagus and brain. Numer-

ous inclusions were present in the surrounding tissue cells, and also in the nuclei of endothelial cells of blood vessels, their presence suggesting a blood-borne ætiological agent. *Herpes simplex* virus was recovered from the liver of one of these babies. Vulval pruritus had been experienced during their pregnancy by two mothers. (*b*) The second group consisted of three older infants with herpetic gingivo-stomatitis, who at autopsy were found to have disseminated herpetic lesions in viscera, including liver, kidneys and œsophagus.

France and Wilmers (1953) record the deaths of twin babies at seven and sixteen days. No history of *herpes labialis* was obtained from the mother, but three days prior to delivery she developed generalized urticaria. These babies were born prematurely; one was well until the fourth day of life, when jaundice became apparent; the other baby was well for two days, when lethargy and weight loss were noted. This latter baby did not became jaundiced, but passed blood *per rectum* prior to death. Autopsy revealed the presence of liver necrosis with numerous nuclear inclusions in both babies. Encephalitis was an accompanying lesion in one case. No virus was isolated from these babies, but the histological appearances suggested *herpes simplex* infection.

The frequency with which viræmia accompanies primary infection is uncertain. Buddingh *et alii* (1953), in a series of twelve children aged from one to three years suffering from herpes stomatitis, failed to isolate the virus from blood, although it was recovered from mouth washings and from fæces. As these children were not examined until the third day of their illness, these authors remark that their results do not justify the conclusion that viræmia does not occur at any time during the disease or during the period of its incubation in the patient. That viræmia may occur is demonstrated by the findings of Ruchman and Dodd (1950), and supported by the second group described by Zuelzer and Stulberg (1952), in which herpetic stomatitis was accompanied by visceral lesions. In the babies who have died from hepatitis and in whom other viscera have been affected, it is reasonable to assume that the virus is spread via the blood-stream. Zuelzer and Stulberg demonstrated in some of their cases the presence of typical nuclear inclusions in the endothelial cells of blood vessels.

The portal of entry of virus into the body is not obvious in the majority of these babies. Herpetic conjunctivitis was present in three of the cases recorded by Zuelzer, whilst skin lesions were present in one, and also in the case recorded by Quilligan and Wilson (1951). In one of Zuelzer's cases, in the twin babies described by France and Wilmers, and also in our two cases there was no lesion of skin or conjunctiva.

One common feature in all cases in which the œsophagus was examined histologically was the presence of herpetic œsophagitis. The most probable time of infection appears to be during the passage of the baby through the birth canal, when contact with herpes of the maternal genitalia could cause skin lesions and conjunctivitis, and inhalation of infected material would result in lesions of the upper part of the alimentary tract.

A history of maternal *herpes labialis* or of *pruritus vulvæ* was obtained in three of the recorded cases, but was not present in either of ours. The absence of a history of maternal herpes in such a high proportion of cases suggests the possibility of infection from other persons concerned in the handling of the newborn.

The case histories presented illustrate an uncommon but clinically recognizable syndrome. The macroscopic appearance of the liver at autopsy and the typical inclusion bodies seen in liver cells on microscopic examination should remove this disease from the nebulous group of neonatal liver necroses. Histological study of other organs and virus isolation will confirm the diagnosis.

Summary.

The clinical histories and morbid anatomical findings of two babies who died from disseminated visceral lesions due to *herpes simplex* virus are recorded. In one case the virus was recovered from tissue obtained at necropsy, and details of its isolation are briefly mentioned.

With the inclusion of these two cases, the literature now presents a total of eleven babies that have died with similar clinical and morbid anatomical changes. On three occasions the virus has been isolated from tissue obtained at necropsy. The typical appearance of the liver should lead to the confirmation of the diagnosis by recovery of *herpes simplex* virus.

Acknowledgements.

Dr. John H. Colebatch kindly supplied the clinical details of Case II. The photomicrographs were reproduced by Mr. E. Matthaei, Faculty Workshops, University of Melbourne. Our thanks are due to Dr. S. Fazekas de St. Groth, who initiated the virus laboratory in this hospital.

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Addendum.

After this paper had been prepared for publication, an article entitled "Hepatic Necrosis in Disseminated Herpes Simplex" was published in *Archives of Disease in Childhood* (1954), Volume 29, page 60. The authors, R. C. B. Pugh, G. H. Newns and J. A. Dudgeon, record the isolation of *herpes simplex* virus from the liver of a baby who died aged forty-two days.

Legends to Illustrations.

FIGURE II.—Photomicrograph of liver (Case I), showing area of necrosis. (Hæmotoxylin and eosin stain, × 85.)

FIGURES III and IV.—Photomicrographs of liver cells (Case II), in which intranuclear inclusion bodies are present. (Hæmotoxylin and eosin stain, × 2000.)

FIGURE V.—Photomicrograph of area of necrosis in adrenal cortex (Case I). (Hæmotoxylin and eosin stain, × 220.)

FIGURE VI.—Photomicrograph of liver (Case II), showing areas of necrosis involving walls of blood vessels. (Hæmotoxylin and eosin stain, × 85.)

FIGURE VII.—Photomicrograph of œsophagus (Case II), showing desquamation of mucosa and inflammation of œsophageal wall. (Hæmotoxylin and eosin stain, × 220.)

FIGURE VIII.—Photomicrograph of lung (Case II), showing mononuclear cells in alveoli. (Hæmotoxylin and eosin stain, × 220.)

A MALIGNANT TUMOUR OF NON-CHROMAFFIN PARAGANGLIA.

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AND
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IN recent years there have been described a number of tumours, which occur in the skeletal muscles mainly of the extremities, and which can be segregated by their clinical behaviour and histological characteristics. Smetana and Scott (1951) presented 14 cases and classified them as "malignant tumours of non-chromaffin paraganglia". Christopherson, Foote and Stewart (1952) describe 12 such cases under the name of "alveolar soft-part sarcoma" and state that the histogenesis is uncertain.

The case to be presented appears to belong to this group.

Clinical Record.

A man, aged forty-two years, by occupation a telegraphist, stated that seven years previously he had knocked his leg at work. A month later he noticed a swelling on the inner aspect of the left thigh (Figure I). Since then it had not increased much in size, but had recently caused occasional sharp radiating pain of saphenous distribution, particularly when it was stretched by muscle action. On the lower and inner aspect of the left thigh, overlying the *vastus medialis*, was a firm, smooth, round mass the size of a large grapefruit. It was mobile in both directions until contraction of the *vastus medialis* fixed it to this muscle. There were some telangiectases in the skin over it, and it was warmer than the same area on the other leg. There were a number of localized tender areas on its surface. General examination of the patient revealed no further abnormality.

At operation this mass, exposed through a curved incision, was found to be mainly encapsulated, but was arising from and adherent to the quadriceps muscle and its expansion, from which it was removed by liberal excision of the medialis muscle. The saphenous nerve crossed the outer surface of the tumour and was adherent to it, so that on superficial examination it appeared to arise from this nerve, which was therefore divided above and below the tumour and removed with it. The ends of the medialis muscle were then approximated with silk and the wound was closed. Healing of the wound was delayed, and a sinus developed, which discharged mainly serous material. The failure of this sinus to respond to conservative treatment, together with the presence of glands in the groin and loss of weight, suggested the possibility of local malignant recurrence. Smears were therefore prepared from the discharge and examined for malignant cells, with negative results. Local exploration was undertaken, but did not reveal the cause of the persistent sinus. The answer came later when a piece of buried silk gave rise to a pyogenic abscess. After drainage of the abscess and removal of the foreign body, healing of the wound finally took place.

Loss of a major portion of the *vastus medialis* prevented full knee extension for some time after operation, but reeducation subsequently overcame this, and now no disability exists.

The operative site was treated with deep X rays. Several chest X-ray films taken have shown no evidence of metastasis.

Macroscopic Description of the Tumour.

The tumour was solid, well defined and encapsulated, measuring seven by six by five centimetres. It was attached to muscle on two-thirds of its surface, with the long axis of the tumour lying in the direction of the muscle fibres. When sections were cut, a sharp line of demarcation was noted between the muscle and the tumour. The cut surface was mottled yellow in colour. There were a few small irregular hæmorrhagic areas (Figure II).

Microscopic Description.

Under low power magnification the tumour was seen to be surrounded by an irregular band of fibrous tissue, which was invaded by masses of tumour cells. Groups of degenerating muscle fibres were present in the outer part of this pseudo-capsule, from which bands of hyaline fibrous tissue proceded into the tumour, dividing it into lobules.

More detailed examination showed the tumour to consist of groups of from 15 to 200 cells separated from one another by thin-walled capillaries. When cut longitudinally these groups appeared as tubular strands. The cells in these groups and strands were often arranged in a glandular, loose fashion at the periphery, an irregular central space being left (Figure III). The arrangement

FIGURE I.

A photograph of the patient taken soon after he first noticed the swelling on the inner aspect of his left thigh.

of the cells was therefore mainly alveolar, with some cords and tubules of cells. This is the most striking feature of the tumour and is often referred to as "organoid" or "endocrine".

Under high-power magnification (Figure IV), the cells were seen to be round, oval or polyhedral in shape, and measured from 15 to 20μ across their widest diameter. The cell membrane was, in most cases, moderately well defined. The cytoplasm was acidophilic, usually with a central clear area, and finely granular at the periphery, and in some cases containing large vacuoles. Some of the cells contained large oval or rod-shaped basophilic bodies. The nuclei were round or oval and measured 5 to 12μ in diameter. They had a finely reticular chromatin arrangement and usually contained one dense well-defined nucleolus. Some nuclei were small and pyknotic, others were large and irregular. An occasional hyperchromatic form was seen. An occasional large multinucleated cell was present.

Tufts of tumour cells were seen bulging into the lumen of some of the blood vessels. Others were invaded by masses of tumour cells (Figure V).

One area was noted where the tumour appeared less well differentiated, the cells being larger and pleomorphic, and the alveoli ill defined (Figure VI). Many of the nuclei in this area possessed large vacuoles, which in some cases pushed the nucleoli to one side.

Reticulum stains showed the groups of cells to be surrounded by reticulum fibres which did not penetrate between the individual cells (Figure VII).

With Masson's trichrome stain the nuclei were dark brown and the cytoplasm was reddish-brown to light brown.

Figure II.
The tumour after having been incised.

There was an occasional cell in which the central part of the cytoplasm stained red. The cytoplasmic bodies were present as dark brown structures. This stain also demonstrated connective tissue around the cords, but not between the individual cells.

With Lendrum's lissamine fast red modification of Mallory's stain, the nuclei stained dark brown and the cytoplasm reddish-brown to light brown.

With periodic acid-Schiff stains, the cytoplasm of the tumour cells stained light brown with some pink areas.

"Scarlet R" stains for fat gave negative results, nor were doubly refractile substances demonstrated.

Discussion.

The tumour presented by us appears to be identical with the series of Christopherson, Foote and Stewart, and with those of Smetana and Scott. The cases described by these authors possess certain distinguishing features. The tumours always appear in association with skeletal muscle, most often in the extremities and usually in young adults. The rate of growth is variable and is sometimes very slow. In some cases many years may elapse from the onset of a noticeable tumour until the time when the patient seeks medical advice. Metastasis in these cases is usually late and occurs most commonly in the lungs. The tumours show certain very characteristic histological appearances.

In the present case there was a long history (seven years), and as the accompanying photograph (Figure I) shows, the patient's observations were accurate when he denied any great increase in size of the tumour after the first month, as this was taken soon after he first noticed it. There was a history of trauma, a common feature with these tumours, which may or may not be of significance. The tumour was in the main circumscribed and apparently encapsulated, the ease of removal making the possibility of recurrence seem remote. However, in six of the cases referred to above local recurrence developed. In this case a large block of muscle was removed with the tumour.

Just over a year has now elapsed since excision of the tumour, but reference to the limited literature on the subject of this tumour shows that a considerable percentage of patients develop secondary deposits in the lungs, some after many years of freedom. One patient from the series of Christopherson *et alii* developed pulmonary metastases after fourteen years.

In view of the liability of these tumours to recur, preoperative irradiation and subsequent amputation would have been better treatment. It is of interest that all cases reported by Christopherson *et alii* except one were dealt with in a similar way by local excision, three of the 12 patients having irradiation. Similarly all but one of the patients of Smetana and Scott were treated by local excision, and three had irradiation.

The histological appearances of these tumours are both striking and distinctive. Their well-marked organoid pattern and the characteristic arrangement of capillaries throughout their substance distinguish them from other neoplasms occurring in this region. They closely resemble the tumours arising from the carotid body, the *glomus jugulare* and other non-chromaffin paraganglia. This, together with the demonstration of paraganglion-like structures in the region of the femoral vessels of the normal thigh, led Smetana and Scott to class them as "tumours of non-chromaffin paraganglia". Christopherson *et alii* expressed no opinion on their histogenesis.

These tumours, although rare, are not so infrequent as the small number of published cases would seem to indicate. Many have been misdiagnosed and have been reported under a variety of headings, the most common being granular cell myoblastoma.

Summary.

A tumour measuring seven by six by five centimetres, present for seven years, was removed from the thigh muscles of a male, aged forty-two years. It was moderately well defined and microscopically conformed to what have been described in the literature as tumours of non-chromaffin paraganglia.

Acknowledgements.

We wish to thank Dr. Malcolm Fowler for advice and criticism during the preparation of this paper. We also thank Mr. B. Fuller for the photomicrographs, and Mr. M. Hansen for preparation of the sections.

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Legends to Illustrations.

Figure III.—A characteristic area of the tumour, showing the alveolar arrangement of the cells. (Hæmatoxylin and eosin stain, × 185.)

Figure IV.—High-power magnification showing the appearances of the tumour cells. The thin-walled capillaries surrounding the groups of cells can also be seen. (Hæmatoxylin and eosin stain, × 370.)

Figure V.—A blood vessel invaded by tumour cells. (Hæmatoxylin and eosin stain, × 185.)

Figure VI.—A less well differentiated area of the tumour showing the poorly defined alveoli. (Hæmatoxylin and eosin stain, × 185.)

Figure VII.—A low-power view of the tumour showing the arrangement of reticulum in the growth. (Krajian silver stain. × 90.)

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A REPORT ON THE CARE OF DEPRIVED CHILDREN.

In Great Britain a good deal of consideration has been given to the care of deprived children—the deserted and homeless, the victims of cruelty and neglect, the maladjusted and the delinquent. In 1944 the Council of the Caldecott Community, at the instigation of its director, Miss Leila Rendel, suggested that the Nuffield Foundation should finance a pilot experiment in the reception, investigation and disposal of deprived children. This proposal received the earnest support of the late Sir Farquhar Buzzard. In 1946 His Majesty's Stationery Office issued what is known as the "Curtis Report"; its proper title was "Report of the Care of Children Committee". Commenting on this report at the time of its issue, *The Times* (London) stated that the guiding principle of the recommendations of the report was the consistent emphasis on "the extreme seriousness of taking a child away from even an indifferent home". It concluded that the proposals of the report would stand or fall, as they should, by its fundamental plea for the concentration of the direction of policy in one department at the centre and of its execution in the local authority, acting through a special committee or a single specialist officer. One of the results of this report was the passage of *The Children Act* (1948), which imposed on every county authority the responsibility of providing at least one centre such as was described in the report. In 1951 the Home Office issued a memorandum in which it was stated that in order to obtain the fullest possible knowledge and understanding of a child's health, personality, conduct, intellectual capacity, emotional state and social history, provision should be made for his reception and temporary accommodation in a place where facilities were available for inquiry into such matters and for observation by a skilled staff. Consequently between October, 1947, and July, 1950, 500 children living in Kent were taken into the Mersham Reception Centre. The background of these children, the homes and the families they came from, their behaviour, their personality, the measures taken to help them, and the outcome during the next two years have been the subject of a book by Dr. Hilda Lewis, entitled "Deprived Children".[1]

Of the 500 children received at the Centre, 55% were boys; the children belonged to 363 families. The proportion of families in the lowest social class was higher than the proportion in the general population, and the fertility of these families was higher than that of the general population. Their incomes and occupational stability were, on the whole, low. Of the families, 30% were living in overcrowded houses; in contrast to this the comparable figure for the general population of Kent was 1·09%. Many of the children's parents were dead, unknown or out of reach. The relationship between the parents who were living together was harmonious in 10% of cases, tolerable in 45% and bad in 45%. Cruelty, drunkenness, crime, indifference to the children and sexual laxity were noted among some of the parents. Many parents had medical disabilities; 99 fathers and 180 mothers exhibited some mental disability or a definite mental illness. Of the children 33% were illegitimate, roughly four times as high a proportion as among the general population. Half the children had been removed from their homes on some previous occasion by a public authority; 63% had been separated from their mothers as a result of wartime evacuation or for other causes; in 28% of cases this separation had lasted for several years. The children had suffered from many changes of school as well as of home. These educational disabilities were shared by most other children in Britain.

The behaviour of the children was, as might be expected, varied. A quarter of them were normal in behaviour and in general mental condition; 25% showed slight and 18% showed severe neurotic symptoms; 22% were delinquent in some degree and 4% were either psychotic or psychopathic. Similarly, the intelligence quotient of 28% of children was below 90; nearly 40% were of average intelligence and 23% had an intelligence quotient above 110. Poor physical condition was manifested by 21% of the children and 38% were underweight. The conspicuous symptoms or characteristics of the children before admission to the Centre were anxiety, enuresis, pilfering and wandering. The most interesting part of this report deals with the influence of the family and environment on the behaviour of the children; this is, of course, of the most practical importance. The matter is not simple, and we read that it would be foolhardy to expect from the study of these children's life that simple causes could be discovered to account for their mode of behaviour. There is no substitute for an affectionate and stable parent, but because in very many instances parents were lacking in desirable qualities the behaviour of the children could not be attributed to this lack. It was possible to see only broad

[1] "Deprived Children: The Mersham Experiment, A Social and Clinical Study", by Hilda Lewis, M.D., M.R.C.P., with a foreword by Arthur Ellis and Dr. C. P. Blacker; 1954. London: Oxford University Press. Melbourne: Oxford University Press. Published for the Nuffield Foundation by Geoffrey Cumberlege. 8¾" × 6", pp. 180. Price: 17s. 9d.

connexions, suggesting rather than demonstrating that particular adverse influences might bring about characteristic deviations of conduct. Hereditary influences were no doubt at work—probably their influence was great—but the inquiry could not throw light on them because the data were insufficient. Large "lacunæ" were found, especially with illegitimate children. It was, in fact, impossible in most instances to sort out the genetic from the environmental and psychological influences supplied by the parents. It would be very simple to attribute findings to the environmental conditions which have been described, but this does not mean, we are reminded, that the hereditary determinants are negligible. "It would be indeed strange if so many maladjusted, dull, neurotic and psychopathic parents as are included in this series did not hand on some of their defects to their children." An analysis was made of 19 features of each child's parental and personal background in order to discover, if possible, a relationship between the child and the normality or otherwise of his mental state when he was admitted to the Centre. Eleven features showed a significant association with mental health. They were maternal neglect, lack of maternal affection, maternal over-indulgence, maternal dulness or mental defect, maternal insanity or other psychopathy, paternal neglect, paternal over-indulgence, separation of the child from the mother before the age of five, prolonged public care and a dirty home. Obviously, many of these features would overlap. The associations were not always those that might have been expected. For example, the group of children from very dirty homes included a higher proportion whose mental state was normal than was found in children from clean homes. A similar disparity in favour of neglected children was evident when children whose mothers had grossly neglected them were compared with those who had attentive mothers. The disparity between children whose mothers were intellectually dull or defective and the others was of the same order. In regard to the pattern of disturbed behaviour, it was found that five background features had a statistically significant association. They were a neurotic or psychopathic mother, a mother lacking in affection for the child, a father lacking in affection for the child, a prolonged stay in public care and illegitimacy. A mentally unhealthy mother tended to produce a neurotic reaction in the child; if the mother or father was not affectionate, or if the child had been in public care, unsocialized aggressive behaviour was more likely to be developed. It was found that unless the separation of the child from the mother had occurred before the age of two years and had been lasting, it bore no statistically significant relation to the normality or otherwise of the child's mental state at the time of admission to the Centre. No clear connexion was evident between separation from the mother and a particular pattern of disturbed behaviour. Problem families have already been mentioned. In these families, the relations between the parents were unsatisfactory in all but six instances, and many of the parents were mentally handicapped in personality or intelligence. The mothers, however, were affectionate to their children. Collectively, the children were of lower intelligence, though they were freer from delinquency and neurosis than the rest of the Mersham children.

At this stage, we may perhaps describe the manner in which the children were placed, though this is set out in the opening part of the book. The recommendations in regard to the children were acted on in 381 instances and were not acted on in 119. The recommendation in regard to the actual placement is set out in a table. Of 42 children who were recommended for return to parents, 38 did return and four were sent to a children's home. A foster home was recommended for 104; eight of these returned to their parents, 73 went to a foster home, two to boarding school, 17 to a children's home and four to an adjustment hostel. A boarding school was recommended for 43 children; 31 were sent to a boarding school, one was returned to the parent, three went to foster homes, six to a children's home, and two to an adjustment hostel. A children's home was recommended for 189 children; 165 were sent to such a home, nine were returned to parents, nine went to a foster home, three to a boarding school and three to an adjustment hostel. The figures for the other categories need not be given in detail, but we may point out that 29 children were recommended for an adjustment hostel, 49 for a school for maladjustment, 13 for a school for the education of the subnormal, 12 for an approved school, and there were 19 in a miscellaneous group. In all, 72 children were returned to their parents, 90 went to a foster home, 38 to boarding school, 209 to children's homes and 91 to adjustment hostels.

One naturally asks what happened to these children, what was the result of their reception and care in such a Centre as that described at Mersham. A follow-up was undertaken of 240 children. These comprised all those who had left the Mersham Centre at least two years before; in some instances, the interval was three and a half years. Two methods of inquiry were used. One was an inquiry by post about the 240 children, and an additional special inquiry was made by a personal visit of 50 boys and 50 girls undertaken by Dr. Hilda Lewis. The reports received by post and the findings obtained by a personal visit and examination of the 100 children tallied in 63% of cases; in a further 23% discrepancies were either trifling or due to the occurrence of an interval between the two inquiries. In the remaining 14%, the discrepancy was definite, though not particularly wide; it was due, in all but one instance, to "an unduly rosy picture in the postal report". It was found that the proportion of children in good psychological and social condition more than doubled during the two years or more since the children were at the Centre. Improvement was shown in varying degree by 63% of the children; no change was noted in 23%, and in 8% the condition of the child was worse. Neurotic symptoms persisted to a greater extent than delinquent behaviour. Of most significance is the finding that children who had been placed in accordance with recommendations made at the Centre fared somewhat better than those who were not placed as recommended. This was also true in regard to such matters as schooling, contact with parents, occupation and holiday arrangements. The general conclusion was formed that the closer the child's contact was with his parents or with near relatives, the better was his condition. If a mother was normally affectionate, this was a good prognostic feature; so also, separation from the mother before the

child was five years of age indicated a bad prognosis. At the same time, nearly a third of the children who were separated from their mothers were in a satisfactory condition at the end of the follow-up period.

Consideration of this report naturally raises the question of the value of such reception centres. Dr. Lewis writes that experience and evidence which have accumulated at the Mersham Centre compel the firm conviction that reception centres, though not ideal, are indispensable at the present time for dealing with a large proportion of deprived children. The point about the reception centres is that they would enable a full and accurate diagnosis of the child's condition to be made. In any ordinary illness, a medical practitioner is handicapped if his diagnosis is not complete; indeed, his treatment may be entirely wrong. When we are dealing with deprived children we are dealing with a problem in social medicine. In clinical medicine, successful treatment will depend, in a large measure, on the persons who have charge of the patient when he is at hospital or at home, not only on their skill but on their outlook and on the way in which they handle the patient generally. In a problem of social medicine such as that exemplified in a reception centre for deprived children, success will depend very largely on the understanding and general suitability of the person in charge. Every necessary diagnostic and therapeutic agent must be available—agents that have to do with bodily, mental and spiritual welfare. It is not conceivable that at the present time anyone like Mr. Bumble, in charge of the orphanage in "Oliver Twist", would be extant, but there are all degrees of unsuitability. If suitability of staff is made the prime consideration, it is safe to conclude that what has been demonstrated at Mersham will be of the utmost value in the care of children who have been deprived of a normal and healthy child life. Finally, this investigation reported by Dr. Hilda Lewis may with advantage be studied along with the report of the investigation of 1000 families in Newcastle-on-Tyne, described in these columns in a recent issue.

Current Comment.

GALEN AS A DIAGNOSTICIAN.

EVEN those members of the medical profession who have no special interest in the history of medicine are familiar with the name of Galen, who lived from about A.D. 130 to A.D. 200, and whose teaching dominated medicine for 1200 years. He is known to have been a conscientious experimenter and careful observer, who recorded what he saw. His anatomical and physiological investigations were revolutionary; his descriptions of physical phenomena arouse admiration today; but some of his deductions from observed facts were much astray. However, we are not concerned in this place with Galen's good or bad influence on the art and science of medicine, but with his clinical acumen. Guthrie[1] gives a good example of Galen's skill as a diagnostician in the following incident. Eudamus the philosopher complained of a loss of sensation in the fourth and fifth fingers of one hand; local treatment by other physicians had failed to effect a cure. Galen, on inquiry, found that his patient had recently fallen from a chariot and struck his neck against a sharp stone. From his knowledge of physiology, he deduced that the brachial plexus was the seat of the trouble. He applied counter-irritants to the region and achieved a cure. Guthrie offers the following pertinent comment: "This success is related by Galen in no modest fashion, for he was a forceful and opinionative man, and his case records differ vastly from the plain and unboastful histories of Hippocrates." That modesty was by no means Galen's besetting sin is well known. It is further exemplified in his own account of a piece of diagnostic detection, which has been set out in a short article by E. Rist.[1] The anecdote, which Galen tells in his work "Of the Affected Parts", is repeated in a free translation of his own words, and may well be retold here. He had been trying to show which symptoms indicated that some particular organ was affected; this incident refers to diseases of the liver.

One day, during his first sojourn in Rome, Galen was met by Glaucus, a philosopher, who begged him to accompany him to the home of a sick Sicilian physician. "Yesterday", said Glaucus, "Gorgias and Apelas told me that you had made diagnoses and prognoses that seemed more like divination than medical art. I should like to have proof, not so much of your learning as of the power of the art of medicine, and to be convinced that it can provide just as startling diagnoses and prognoses." By this time they had arrived at the sick man's home. At the front door they met a manservant, who was taking away from the sick-room some "soapsud" fæces containing thin, blood-stained pus; Galen regarded this type of stool as invariably indicating liver disease. He let it appear that he had seen nothing, and accompanied Glaucus to the bedside of the patient; there he "seized the patient's arm, wishing to find out whether inflammation was present, or only atony of the organ". Rist points out that this simply means that Galen took the patient's pulse, the fast rate of which indicated fever. The patient, who was a physician himself, volunteered the information that he had just been to stool, and that his pulse rate was faster because of the effort. However, Galen regarded it as indicating inflammation. Looking about him, he observed on the window-sill a pot containing a preparation of *Hyssopus officinalis* and honey water; he deduced from this that his colleague considered himself to be suffering from pleurisy, because he felt pain at the level of the false ribs; Galen points out that such pain sometimes accompanies inflammation of the liver. Galen, pursuing his deductions, gathered from this that the patient's respirations would be rapid and short, and that he was likely to be troubled by frequent attacks of coughing. The medical detective was elated to find that he had an opportunity of increasing the admiration in which he was held by Glaucus, and, placing his hand on the false ribs of the patient, said that he had pain in that area. The sick man agreed, and Glaucus, thinking that the patient's pulse had been sufficient to give Galen that information, could not conceal his admiration. In order to cause him still more astonishment, Galen told the patient that no doubt he felt the need to cough, and that at fairly long intervals he had a little dry cough, with no sputum. As he spoke, the patient by chance coughed exactly in the manner described. Thereupon Glaucus was amazed, and began to shower praises upon Galen. "Do not think", Galen said to him, "that those are the only things that the art can divine about sick people. There are others which I shall show you, and which the patient himself will confirm." Turning to the patient, Galen suggested to him that when he breathed more deeply he felt a sharper pain in the area indicated, and at the same time a sensation of weight in the right hypochondrium. At these words the doctor-patient himself was astonished, and joined his praises to those of Glaucus. Galen, recognizing the success he was having, was tempted to try another suggestion about the sensation of dragging on the clavicle; but although he knew that it accompanied serious liver diseases like cirrhosis, he did not want to run the risk of detracting from the admiration that he had aroused. He decided to slip the suggestion in carefully. "Soon", he said to the patient, "you will feel a sensation of dragging on the clavicle, if you have not already felt it." The astounded patient agreed that he

[1] Guthrie, D. (1945), "A History of Medicine", Nelson, 76.

[1] *Presse méd.*, December 15, 1954.

had already felt that sensation. To complete the bewilderment of his colleague and Glaucus, Galen then said that he proposed to tell them the patient's opinion about his own illness; both thought it likely that he could do so. When he said that the patient thought himself to be suffering from pleurisy, the chorus of admiration was augmented by the manservant, who had just given him an affusion of oil on that account. Galen concludes his story as follows: "Glaucus thenceforth held a high opinion of me and of the art of medicine, which previously he had held in low esteem, never having made the acquaintance of men who were outstanding in the practice of the art."

Rist points out that the story is vivid and picturesque; but Galen was satisfied with having dumbfounded Glaucus, the sick doctor and the reader. It would be nice to know what happened to the patient, but that we are not told. Criticism may be levelled at one of Galen's deductions; Rist points out that the "soapsud" stools that Galen describes are indeed a sign of liver disease; they are described in all the classical text-books. However, now they are held to be a characteristic symptom of acute dysentery. Rist puts forward the suggestion that the patient may have been suffering from amœbiasis of the colon, and perhaps from an incipient amœbic abscess of the liver. The ancients had no suspicion of such a causal connexion, which was shown to exist only as recently as the last century. Whatever one may think of his clinical acumen, Galen's examination of the patient was incomplete, superficial and elementary. As a piece of medical detection, however, the anecdote is intriguing, and we are led to think of Sherlock Holmes and Dr. John Thorndyke; we may even find some similarities of personal characteristics between the former and Galen, who has been called the greatest doctor of antiquity.

TRENDS IN THERAPEUTIC ABORTION.

ETHICAL CONSIDERATIONS make controversy about therapeutic abortion difficult to resolve, but the situation is being made easier in other ways. As R. Vernon Colpitts[1] has pointed out, advances in medical management and newer concepts of disease processes have tended to reduce the number of indications for therapeutic abortion, some to the vanishing point. In discussing the changes in indications over the years he considers them in seven groups: toxæmia, cardiac disease, pulmonary disease, urological disease, neurological and psychiatric disease, medical diseases and miscellaneous conditions. Toxæmia, including renal and hypertensive disease, constitutes the largest single indication for therapeutic abortion. Evidence of disease is usually demonstrated by persistent albuminuria and impairment of renal function. If nephritis is present at the time of conception, Colpitt's view is that abortion should be performed promptly. If the disorder is latent and arises early in pregnancy and if it is marked by albuminuria which increases despite treatment, it is unlikely that pregnancy will succeed. If to albuminuria are added œdema and hypertension, abortion becomes obligatory.

Therapeutic abortion for cardiac disease is declining and has almost vanished in some clinics; in particular, advances in cardiac surgery have been responsible for decreased therapeutic abortion and increased fœtal salvage. Contradictory views are held by different authorities on therapeutic abortion for pulmonary tuberculosis. Recent surveys show that women with pulmonary tuberculosis do just as well when pregnancy is allowed to continue as when it is therapeutically interrupted. Fewer therapeutic abortions are now performed for urological diseases. Active renal tuberculosis is considered an indication for termination of pregnancy, but malignant disease of the urinary tract is not considered an indication.

Turning to neurological and psychiatric diseases complicated by pregnancy, Colpitts states that in the past two decades the number of cases necessitating abortion has dropped, while the number of cases in which it may be done has increased. In most psychoses therapy may be employed during the course of pregnancy without adversely affecting the mother or the baby. Medical conditions such as hyperthyroidism, ulcerative colitis, Raynaud's disease and sarcoidosis are no longer considered valid indications for therapeutic abortion. The risks of the "rubella syndrome" when rubella complicates early pregnancy are considered high by Colpitts. In 199 cases, only 32 (16%) of the children were born without defects; 100% were defective when the disease occurred in the first month of pregnancy. Colpitts considers abortion to be indicated in women already immunized from preceding pregnancies which had resulted in one or more erythroblastotic infants with fatal forms of the disease, when their husbands' blood groups were such as to exclude the possibility of an Rh-negative infant.

No doubt these indications can be further whittled down or even eliminated by those to whose moral sense any form of abortion is intolerable; but at least it is good to learn of some easing in one of the paradoxical situations of medicine, when one life has to be balanced against another.

[1] *Am. J. Obst. & Gynec.*, October, 1954.

SELENIUM SULPHIDE SHAMPOO FOR SEBORRHŒIC DERMATITIS.

SELENIUM is close to sulphur in the periodic tables, and because of this and its general properties it is not surprising that its trial has been considered for skin therapy, a field in which the usefulness of sulphur has long been recognized. On the other hand, the toxic properties of selenium have been known and described in the literature for over a century, and before W. N. Slinger and D. M. Hubbard[1] were prepared to submit it to clinical trial they investigated its toxic properties with great care, first in animals and then in human volunteers. Their fears were quickly allayed. It was found by forty-eight-hour and repetitive patch tests for four weeks that selenium disulphide used in a shampoo produced neither primary irritation nor sensitization reactions in 100 normal subjects. The shampoo containing selenium disulphide was then used therapeutically over periods from one to seven months in 104 cases, 90 of which were cases of seborrhœic dermatitis, without consequent signs of irritation, sensitization or toxicity. The shampoo completely controlled 81% of the cases of all degrees of severity, 95% of the mild cases and 85% of the moderate cases. It exerted no influence on the clinical course of seven cases of *tinea capitis* associated with the presence of *Microsporon audouini*. It produced benefit in two cases of atopic dermatitis and three cases of exfoliative dermatitis in that the amount of scaling of the scalp was reduced. The use of the shampoo on the diseased scalp of each of 19 patients did not result in the absorption of selenium to an extent that could be detected on the basis of any regular or significant increase in the excretion of selenium in the urine. Moreover, the use of the shampoo on badly fissured scalps of two patients for fourteen and nineteen successive days respectively failed to produce definite evidence of initial or progressive absorption of selenium. Not long after the appearance of this report, A. H. Siepyan[2] reported control of seborrhœic dermatitis of the scalp in 87% of a series of 286 patients treated with selenium disulphide shampoo. Simple dandruff or *pityriasis sicca* responded in 92% of cases, and *pityriasis steatoides* was controlled in 85% of cases. Psoriasiform seborrhœa was alleviated but not cleared without additional local therapy. The itching due to *seborrhœa oleosa* was controlled, but the oiliness of the hair was exaggerated. Patients with *acne vulgaris*, *acne juvenilis* and atopic eczema of the scalp received some benefit. No sign of intoxication, sensitization or irritation was noted over an eight-month period.

[1] *Arch. Dermat. & Syph.*, July, 1951.

[2] *Arch. Dermat. & Syph.*, February, 1952.

A number of subsequent investigators have agreed with these findings, and in a recent paper E. S. Bereston[1] was able to report a study of 140 cases extending over two years. He found selenium sulphide shampoo to be a safe, non-toxic, efficient method of controlling seborrhœic dermatitis of the scalp in 89% of his cases. The two objections raised were that 31% of the patients found their hair to be excessively oily after three or four weeks of using the shampoo, and 19% of the patients developed an orange tint of their grey hair. The excessive oiliness is partially corrected by the use of tincture of green soap shampoo before the selenium sulphide shampoo, and the orange tint can be diminished by thorough rinsing, but neither problem is as yet fully solved. Mild conjunctivitis developed in 1% of cases when the shampoo went into patients' eyes, but this cleared in a few days in all cases. The vast majority of patients observed were very satisfied with the treatment because it was successful and simple. It is, of course, particularly acceptable to patients who either do not respond to sulphur, resorcin and other drugs or are sensitive to them. The technique is to wash the scalp every week with any soap or a regular shampoo and to rinse thoroughly. Then when the hair is still wet, two teaspoons of selenium sulphide are applied and thoroughly massaged into the scalp. A foamy lather results shortly afterwards which should be left on the scalp for five minutes before being washed off. Notable improvement is reported to occur within a few weeks, but the use of the shampoo must be continued on a permanent weekly basis to prevent recurrence of seborrhœa. However, Bereston emphasizes the fact that it does not cure the condition.

THE REQUIREMENTS FOR A SURVIVAL RATION.

THE physiological effects of restricted feeding, particularly on protein utilization, have occupied the interest of nutritionists for a long time. The conditions which may be set up in modern warfare have increased the importance of the subject. These conditions include the cutting off from normal supply channels, especially of victims of air and sea disaster and civilian populations in disrupted areas. As an aid in the design of a multipurpose survival ration information relative to normal active men in the military age group has been compiled by Doris H. Calloway and H. Spector, of the Quartermaster Food and Container Institute for the Armed Forces at Chicago, Illinois, United States of America.[2] Emphasis has been placed on the more acute, short-term experiments. Studies were chosen from the literature in which the subjects were young men, essentially normal in weight and nutritional status, who were permitted at least sedentary activity. Data on control groups indicate that the men normally maintained body weights at intakes of 3000 to 3600 Calories per day. Estimates of caloric expenditure during the restriction phase ranged between 2000 and 3500 Calories daily. Great care was taken in selecting the data to ensure that all the relevant information was given. Feeding nitrogen to 6·9 grammes daily was of little benefit when only 400 to 600 Calories were supplied. Protein fed under these circumstances is largely burnt as an energy source with a rise in urinary nitrogen excretion. An intake of 900 Calories was the lowest level at which addition of protein to the diet produced less negative nitrogen balance than the same number of protein-free Calories. A plateau at 1·8 to 2·4 grammes of negative nitrogen balance was reached at approximately 1600 Calories which persisted through approximately 2300 Calories. As 7·0 grammes of total nitrogen excretion is the baseline for protein-free feeding, no increase in the energy content of the diet could promote nitrogen balance when the nitrogen intake was below this amount. At Calorie levels of 400 to 600 dietary nitrogen is without appreciable benefit. When the Calorie intake is approximately 1000, 3·0 grammes of nitrogen may be used to good advantage, but more is without further benefit. At Calorie levels of 1400 to 2300, 7·0 to 9·0 grammes of nitrogen are adequate. When the full Calorie requirement was met 8·5 grammes of nitrogen were sufficient to promote balance and very little if any more was retained.

These data provide the basis for the development of protein-containing survival rations. One of the factors which has to be taken into account is the possibility of shortage of water for drinking and the fact that every gramme of urea nitrogen requires 40 to 60 millilitres of water for excretion. A versatile food unit of 500 Calories, 7% to 8% of which are derived from protein, is suggested. Such a unit could be consumed in any multiple number with maximum benefit from the protein at each energy level. The actual composition of the diet is apparently not critical excepting that sufficient carbohydrate should be provided to prevent ketosis.

[1] *J.A.M.A.*, November 27, 1954.

[2] *Am. J. Clin. Nutrition*, November-December, 1954.

SUBARACHNOID HÆMORRHAGE.

THE ocular signs of subarachnoid hæmorrhage have been a matter for controversy for many years in regard to their frequency and pathogenesis. W. A. Manschot[1] attempts to elucidate the problem. He has reviewed 225 cases of spontaneous subarachnoid hæmorrhage and examined serial sections of globes and the orbital part of the optic nerves of three patients with a spontaneous subarachnoid hæmorrhage who also had intraocular hæmorrhages. He has also examined serial sections of the bony optic canal and its surrounding tissues from three patients with subarachnoid hæmorrhage who were found at post-mortem examination to have a hæmorrhage between the optic nerve sheaths; these had no intraocular hæmorrhages. He injected indian ink into the subarachnoid space to prove the presence or absence of an open communication between the cerebral subarachnoid space and the subarachnoid space around the optic nerves. In contrast to Sir Stewart Duke-Elder's[2] statement that papillœdema is the most constant symptom and that retinal hæmorrhages are present in the majority of cases, Manschot found only 45 patients with intraocular hæmorrhages and only 38 with papillœdema. However, there is agreement that hæmorrhages in the fundi indicate a bad prognosis. The greatest controversy arises in endeavouring to determine the pathogenesis of the intraocular hæmorrhages. F. B. Walsh and T. R. Hedges, junior,[3] A. J. Ballantyne[4] and F. C. Cordes[5] all agree that the blood in the cerebral subarachnoid space does not as a rule travel through the optic foramen into the subarachnoid space of the optic nerve, but that hæmorrhages are explained by a sudden rise in intracranial pressure causing a stasis in all venous channels which drain the tissues of the eye and the contents of the orbit. Manschot is of the opinion that a rise in intracranial pressure could never produce a venous stasis in all the venous channels which drain the orbit. His injection experiments on cadavers, in which indian ink was injected suboccipitally into the subarachnoid space, showed that the ink had traversed the whole length of the subarachnoid space in the optic canal. His conclusions therefore are that intraocular hæmorrhage in patients with spontaneous subarachnoid hæmorrhage is explained by a sudden intracranial hypertension that forces blood and liquor under high pressure into the subarachnoid space of the optic nerve through an open communication in the optic canal. Compression of the central retinal vein where it traverses the intervaginal spaces and compression of the choroidal anastomoses of the central retinal vein cause venous stasis. The congestion is aggravated by increased blood flow through the central retinal artery. These forces are sufficient to cause rupture of retinal veins and capillaries.

[1] *Am. J. Ophth.*, October, 1954.

[2] "Textbook of Ophthalmology", Volume III, page 2939.

[3] *Am. J. Ophth.*, April, 1951.

[4] *Brit. J. Ophth.*, September, 1943.

[5] *Am. J. Ophth.*, September, 1953.

Abstracts from Medical Literature.

MEDICINE.

Aortic Stenosis and the Serum Cholesterol Level.

E. P. BOAS *et alii* (*Am. Heart J.*, October, 1954) found the serum cholesterol level of patients with aortic stenosis to be abnormally high in about one-half of the women and about one-quarter of the men. Among patients with chronic rheumatic valvular disease there was hypercholesterolæmia in about one-fifth to one-seventh of the patients, depending on the standard used, and the incidence in men and women was about equal. Of the patients with aortic stenosis about 70% had had previous rheumatic infection. The authors suggest that in persons with hypercholesterolæmia aortic stenosis may be caused by atherosclerosis of a normal aortic valve, but that in most instances it is caused by atherosclerosis implanted on aortic cusps scarred by former rheumatism.

The Dynamics of Mitral Insufficiency.

S. RODBARD AND F. WILLIAMS (*Am. Heart J.*, October, 1954) have studied by means of models the hydrodynamic factors affecting the regurgitation of blood through the mitral valve. In particular they have examined the problems of pipe competition in relation to the competition between the mitral and aortic openings for streamlines of flow. They have demonstrated that when mitral regurgitation is present, the orifice through which it passes must be very small to be compatible with an adequate flow of blood into the aorta, also that serious errors may arise in attempts to calculate orifice size from pressure and flow relationships. Their findings agree with the observation of strong contraction of the mitral ring at the onset of ventricular systole. The size of the mitral orifice at autopsy bears no relation to the size of the orifice during life.

Chronic Cerebral Hypertensive Disease.

W. HUGHES, M. C. H. DODGSON AND DOROTHY C. MACLENNAN (*Lancet*, October 16, 1954) report a study of 51 hypertensive subjects with clinical evidence of cerebral damage. They state that the incidence in the sexes was about equal. Most of the patients were elderly, but a few were middle-aged. The clinical pattern was characterized by frequent small strokes with transient or permanent paralysis and the tendency to develop pseudobulbar palsy in the course of time. Characteristic personality changes included emotional lability and intellectual deterioration. The disease is stated to be chronic with an observed duration of one and a half to twenty years. It tends to start somewhat earlier and to run a more rapid course in males than in females. Those who survive long tend to drift into dementia. Lesions occur mainly in the territories supplied by the perforating arteries. The authors state that the existence of a group of cases such as they describe has not hitherto been recognized, although scattered accounts of individual cases of cerebral arteriosclerosis and malignant hypertension have been published. The limits of the group are defined by finding hypertension in every case, by the development of emotional lability and pseudobulbar palsy, by personality changes and by the tendency to end in dementia. From the authors' study of the pathology so far, it appears that all these profound changes in behaviour and personality derive from lesions in the basal ganglia, while the cerebral cortex remains intact.

The Rheology of Post-Stenotic Dilatation.

E. HOLMAN (*J. Thoracic Surg.*, August, 1954) has studied the phenomenon of post-stenotic dilatation of blood vessels, which is seen distally to subaortic stenosis, to pulmonary stenosis, to pulmonary infundibular stenosis, to coarctation of the aorta and to various other forms of arterial stricture. A mass of fluid ejected through a narrow and limited constriction at high velocity strikes against a more slowly moving mass of fluid distal to the stricture; this results, first, in the conversion of high kinetic energy into high potential energy or lateral pressure and, second, in the lateral deflection of the rapid stream and even in the complete reversal of its direction of flow. This produces a clash of opposing streams and eddies of alternating high and low pressures, whose repeated impacts over a long period against an elastic wall are capable of inducing structural fatigue and distension of that wall.

Tonsils and Poliomyelitis.

G. W. ANDERSON AND J. L. RONDEAU (*J.A.M.A.*, July 24, 1954) discuss the absence of tonsils as a factor in the development of bulbar poliomyelitis. They state that many reports have indicated that bulbar and other forms of poliomyelitis develop more often in patients who have undergone tonsillectomy. The authors studied 2669 cases of poliomyelitis. Of 535 persons with bulbar poliomyelitis 71% gave a history of tonsillectomy. Of persons suffering with mild or severe spinal poliomyelitis and of those with non-paralytic poliomyelitis, only 28% to 34% gave a history of tonsillectomy. Tonsillectomy is said to predispose to poliomyelitis, especially that of the bulbar type.

Salt and Blood Pressure.

J. MCDONOUGH AND C. M. WILHELMJ (*Am. J. Digest. Dis.*, July, 1954) describe the effect of excess salt intake in a healthy young male. For twenty-three days he was under observation. Then for twenty-five days he was given about 40 grammes of salt by mouth daily, in addition to the salt taken at meals. As a result, his blood pressure rose from 120 millimetres of mercury, systolic, and 80 millimetres, diastolic, to 150 millimetres of mercury, systolic, and 100 millimetres, diastolic, and his weight increased from 81 to 84·5 kilograms. When the excess salt was omitted, his blood pressure and weight fell to normal within three or four days. The conclusion was that massive doses of salt, one to two ounces a day in addition to dietary salt, caused an increase of weight and blood pressure. No conclusions are drawn as to the effect of moderate doses of salt.

Osteosclerosis Associated with Chronic Renal Failure.

T. CRAWFORD, C. E. DENT, P. LUCAS, N. H. MARTIN AND J. R. NASSIM (*Lancet*, November 13, 1954) draw attention to the paradoxical finding of osteosclerosis in some previously described cases of chronic renal failure. They report three new cases of the same kind. In only one case was there marked enlargement of the parathyroid glands and typical *osteitis fibrosa* in addition to the osteosclerosis. In the two other cases there were no signs of hyperparathyroidism. The authors state that current knowledge of bone metabolism seems inadequate to explain these findings in renal failure. In two of the cases and possibly in other published cases not remarked on specifically, there were disproportionately long limbs. The authors suggest, with some hesitation, that this is further possible but rare evidence of chronic renal failure occurring before growth of the long bones ceases.

Acoustic Neuroma.

F. A. ELLIOTT AND W. MCKISSOCK (*Lancet*, December 11, 1954) state that the post-operative mortality and morbidity in patients with acoustic neuromata are disappointingly high, especially in view of the benign character and relative accessibility of most of these tumours. Three cases are described in which diagnosis was made at a stage when only the seventh and eighth nerves were involved by the growth. The tumours were removed *in toto* without increasing the pre-operative level of deafness and without causing facial paralysis. The authors emphasize the statement that the diagnostic value of otological tests, especially the loudness balance test, may present evidence to show that depression of the corneal reflex in early cases is due to interference with the motor part of the reflex arc in the facial nerve and does not necessarily imply damage to the trigeminal sensory route. It is suggested that damage to the vestibular component of the eighth nerve may cause unsteadiness of stance and gait, and that these symptoms do not necessarily indicate interference with the cerebellum, though they may complicate the cerebellar ataxia seen in more advanced cases. The possibility is emphasized of early diagnosis at a stage when symptoms are confined to disturbances of eighth nerve function.

Tuberculosis and Coal Workers' Pneumonoconiosis.

A. L. COCHRANE (*Brit. J. Tuberc. & Dis. Chest*, October, 1954) reviews the evidence on the prevalence and on the attack and mortality rates of tuberculosis in coal miners. He suggests that a little coal dust retained in the lungs has a definite therapeutic effect, but that larger accumulations increase the attack and mortality rates from tuberculosis.

He states that the idea that coal dust may have a harmful effect on the lungs is comparatively modern, and there is much evidence to the contrary.

Pleurisy with Effusion.

P. A. EMERSON (*Brit. J. Tuberc. & Dis. Chest*, October, 1954) has found in a series of some 40 patients that treatment with prolonged bed rest did not reduce the incidence of subsequent tuberculosis in patients suffering from pleural effusion. He suggests that some other treatment, such as the use of antibacterial drugs, is necessary to prevent the high subsequent incidence of tuberculosis.

Hypercapnia and Cardiac Arrest.

W. C. SEALY *et alii* (*J. Thoracic Surg.*, November, 1954) produce evidence that disturbances of electrolyte metabolism due to hypercapnia are potent factors in the production of ventricular fibrillation and cardiac arrest. Hypercapnia leads to progressive hyperkalæmia, which increases further immediately after the cessation of hypercapnia, owing, it is thought, to the liberation of adrenaline. Striking changes in the electrocardiogram, usually including bradycardia, give warning of impending ventricular fibrillation before other evidence of impaired circulation occurs. Ventricular fibrillation may be prevented by the intravenous injection of 20% glucose and 3% sodium chloride solution as soon as the warning electrocardiographic changes set in.

Cerebral Vascular Disease.

IRVING S. WRIGHT AND ELLEN MCDEVITT (*Ann. Int. Med.*, October, 1954) discuss cerebral vascular diseases and their significance, diagnosis and present treatment including the selective use of anticoagulant substances. They state that today it is increasingly important to determine whether neurological signs of an intracranial lesion are due to hæmorrhage, local thrombosis, embolism from a fibrillating heart, aneurysm of a cerebral artery or a tumour. If the type of lesion and its location are fortunate, some form of therapy can now be at least logically considered for many patients. The authors present statistics to show that cerebral vascular diseases are among the most serious and most common diseases. Differential diagnosis is no longer of purely academic interest, and various methods to aid in diagnosis are discussed and evaluated. Vasospasm occurs in the cerebral arteries and is of importance in terms of warning of more serious phenomena to come, and also because treatment may be of some help in preventing the serious sequences. Surgical methods may be considered for some subdural or extradural hæmorrhages, but most other cerebral hæmorrhages are not amenable to specific therapy. The use of sympathetic blocks for acute thromboses or emboli is under study at present, but final evaluation of this procedure remains for the future. The use of anticoagulants for the treatment of thrombosis and embolism is also in the process of investigation, and their trial appears justified. Experience with the use of anticoagulants in the reduction of thrombo-embolic episodes in 57 patients treated for a period of 1162 months is summarized. The 57 patients during a period of 795 patient-months before beginning anticoagulant therapy experienced 205 thrombo-embolic episodes, 81 of which were cerebral in location. After institution of anticoagulant therapy, during a period of 1162 patient-months, these patients experienced 23 thrombo-embolic episodes, six of which were cerebral in location. The reduction appears to occur in emboli arising from thrombi in the hearts of patients suffering with rheumatic heart disease or from acute myocardial infarction. A reduction in the incidence of recurrent thrombosis in the arteries of the brain has also been noted. The risk from hæmorrhage in the treatment of these diseases with anticoagulants is present but not excessive.

Coronary Occlusion, Infarction and Diabetes Mellitus.

MAURICE FELDMAN AND MAURICE FELDMAN, JUNIOR (*Am. J. M. Sc.*, July, 1954) discuss the association of coronary occlusion and infarction with *diabetes mellitus* based on a study of 1319 consecutive adult necropsies, in which 137 cases of *diabetes mellitus* were found, that is, 10%. Of these 60, or 43%, revealed evidence of coronary occlusion and infarction. Review of the literature shows that the necropsy incidence of coronary disease associated with *diabetes mellitus* varies from 28% to 52·3%. The greater incidence of this series may be explained by the larger number of Jewish patients involved, as diabetes is more prevalent among this race. The incidence of the association of coronary heart disease with diabetes (43%) is compared with the 20% incidence of coronary occlusion and infarction noted in the authors' study of the necropsy findings in non-diabetic adult subjects. This indicates that coronary occlusion is twice as prevalent in the diabetic as in the non-diabetic. Other observers are generally agreed that the degree of cardiovascular change is greater in diabetic than in non-diabetic patients, and that its onset occurs at an earlier age. In this series, the sex ratio was the same in the diabetic group with, as in that without, associated coronary occlusion. In both groups, there was a decided preponderance of females (60% and 61% respectively). This is in contrast to the predominance of males among patients with coronary disease but without diabetes.

Cardiac Failure in Thoracic Deformities.

J. W. FISCHER AND ROBERT A. DOLEHIDE (*Arch. Int. Med.*, May, 1954) report the clinical and autopsy findings in 11 subjects who died of cardiac failure as the result of deformity of the chest. The cause of the chest deformity was usually not clear. The chest deformity was so great that dwarfism existed. The cardinal symptom was dyspnœa. The disorder may exist for a large part of a lifetime before more acute symptoms lead to a rapid demise. Pulmonary symptoms are usually prominent, and pneumonia, emphysema, bronchitis, bronchiectasis and atelectasis are frequently found alone or in combination at autopsy. The physical findings in the heart are variable and often difficult to elicit. Gallop rhythm has occurred frequently. The chest skiagrams usually reveal depression and atelectasis on the side of the deformity, while the opposite side is the site of compensatory emphysema. The heart is of a "mitral shape". The electrocardiographic pattern is as a rule consistent with a state of *cor pulmonale*. It is of interest that significant microscopic abnormalities in the lung are strikingly lacking. The authors believe that deformity of the chest is frequently not appreciated as an ætiological factor in some cases of cardiac failure. It is thought that both pulmonary and cardiac failure contribute to the clinical picture, but the actual part played by each of these is not at present understood.

Marrow Iron in Anæmia.

PEYTON T. PRATT AND MARLIN E. JOHNSON (*Arch. Int. Med.*, May, 1954) have measured qualitatively the bone marrow iron stores in 128 marrow biopsies. They find increased iron stores in the presence of macrocytic anæmias and of normocytic anæmias not due to acute bleeding. This is noted particularly in the presence of chronic infection. The iron stores are reduced in patients suffering from chronic bleeding and nutritional anæmias of infancy. In pregnancy the hæmoglobin level is possibly related to reduction of iron stores.

Mitral Stenosis in Facsimile.

FRANK W. DAVIS, JUNIOR, AND E. COWLES ANDRUS (*New England J. Med.*, August 19, 1954) report three cases in which a diagnosis of mitral stenosis was made on clinical and other grounds but the patients subsequently proved to have other lesions. In the first case a myxoma of the left auricle was found at operation, while in the second autopsy revealed a congenital anomaly of the myocardium of the left ventricle which was producing effective stenosis in the mitral region. The third patient had a chronic collagenous degeneration which was constricting the pulmonary veins and creating inflow obstruction into the left atrium. In none of these patients was there any satisfactory explanation for the diastolic murmur; none of them had an opening snap. The authors suggest that exploratory cardiotomy is advisable in cases in which obstruction seems to exist, even though a definitive clinical diagnosis is impossible.

Resuscitation after Cardiac Arrest.

L. N. TURK AND W. W. L. GLENN (*New England J. Med.*, November 11, 1954) report 45 cases in which cardiac arrest was diagnosed in one hospital during a period of five years. In two of the patients the heart was found to be beating when the chest was opened, and in one it resumed activity spontaneously. These three patients recovered uneventfully. Resuscitation by cardiac massage and other supportive measures was attempted 42 times. There was complete success in seven instances, 16 patients were temporarily or partly resuscitated, and in 19 cases no cardiac function could be restored.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on July 7, 1954, at Saint Vincent's Hospital, Melbourne. The meeting took the form of a series of clinical demonstrations by the medical and surgical staffs of the hospital.

Surgical Treatment of Carcinoma of the Stomach.

DR. CHARLES OSBORN presented a demonstration of the results of surgical treatment of carcinoma of the stomach. A survey of a series of patients treated by him during the past eight years provided the basis of the discussion. Although the long-term survival rates emphasized the malign nature of the disease, there was sufficient evidence to warrant a somewhat less pessimistic view than that commonly held. In conformity with all other observers, the facts revealed the relatively short history of illness of most patients presenting for examination. Yet in spite of this, it was a constant source of alarm when the advanced stage which the disease had reached was revealed at operation. Furthermore, in the absence of macroscopic evidence of metastatic spread, it was impossible to prognosticate the outcome of radical surgical treatment at the time of operation. Dr. Osborn said that the causes determining that state of affairs appeared to be related to the cell structure combined with other factors at present unknown.

Dr. Osborn then reviewed 49 cases. Ten of the patients were found at operation to have inoperable growths; seven were submitted to palliative resection, of whom three died after operation and four died within one year. Thirty-two patients were submitted to resection and their survival periods were: more than eight years after operation, one patient remained alive; more than seven years after, one patient remained alive; more than six years after, of three patients one had died of tuberculosis and two remained alive; more than four years after, one patient had died; more than three years after, one patient had died; more than two years after, of four patients two had died and two remained alive; more than one year after, of five patients four remained alive and one had died; less than one year after, of 12 patients nine had died and three remained alive; there were three post-operative deaths and one anæsthetic death.

The survival periods of those patients who were submitted to subtotal gastrectomy were compared with those on whom a total gastrectomy was performed. It was not suggested that any conclusion of value could be drawn from the comparison as the series was obviously far too small, and a number of factors, other than the pathological, determined the type of the operation in individual cases.

Dr. Osborn said that there had been 14 partial resections; more than eight years after operation, one patient was alive; more than seven years after, one patient was alive; more than four years after, one patient had died; more than two years after, of two patients one was alive and one had died; more than one year after, of two patients one was alive and one had died; less than one year after, of four patients all had died; post-operative deaths numbered two. Total resections had numbered 18; more than six years after operation, of three patients one had died of tuberculosis and two remained alive; more than two years after, of three patients two were alive and one had died; more than one year after, one patient remained alive; less than one year after, of eight patients three were alive and five had died; post-operative deaths numbered two, and there had been one anæsthetic death.

Dr. Osborn went on to say that the most interesting feature of the series, and perhaps the most significant, was the review of the patients who had been operated on more than five years previously. Of the 12 patients, five had survived for five years or more, one had died after four years from unknown causes, one had died from carcinoma after two and a half years, three had died within one year, and there had been two post-operative deaths. Present at the demonstration were four patients who had survived for more than five years; they all appeared to be in good health and in a satisfactory state of nutrition, and were all leading normal lives for their age. The patients were: a female, aged sixty-eight years, alive eight years after operation; a male, aged sixty-two years, alive seven years after operation; a female, aged seventy years, alive six years after operation; and a male, aged fifty-three years, alive six years after operation. One other patient, a male, had died six years after operation from tuberculosis, in a sanatorium; a necropsy had revealed no evidence of recurrence of carcinoma.

Dr. Osborn also discussed some of the complications encountered after total gastrectomy, including nutritional disturbances, anæmia and stenosis at the œsophago-jejunal anastamosis. He favoured the Roux *en Y* type of anastamosis, as that procedure seemed to eliminate the unpleasant biliary regurgitation that not uncommonly followed the loop anastamosis.

Endocrine Disorders.

DR. W. HAMILTON SMITH showed patients with disorders of the endocrine system.

Simmonds's Disease.

Dr. Hamilton Smith first discussed three patients who had presented with Simmonds's disease (hypopituitarism). The first patient was a woman, aged fifty-eight years, who had been admitted to hospital in a coma after thirty-six hours of dry retching and diarrhœa. Later a history was obtained of ill health and amenorrhœa following an abortion at six weeks, twenty years previously. The patient had felt the cold very badly, was constipated, had been treated for pernicious anæmia for ten years and had not left her bed for two months. On her admission to hospital the patient was in a deep coma and was incontinent of urine and fæces; her temperature was below 96° F. and her blood pressure was not recordable. She had lost a moderate amount of weight and all her axillary and pubic hair; her head hair was thinned and lifeless. The skin was pale and waxy. Glucose (50% solution) given intravenously led to slight improvement. Great improvement followed the exhibition of cortisone, 100 milligrammes by intramuscular injection, followed by 25 milligrammes by mouth. The hæmoglobin value was 65% (9·6 grammes *per centum*), the direct eosinophile cell count was 77 per cubic millimetre, the basal metabolic rate was −30%, the blood cholesterol content was 320 milligrammes per 100 millilitres, and the urinary 17 ketosteroid excretion was three milligrammes per twenty-four hours. Excretion of a water load (Kepler-Power test) was impaired. The electrocardiogram showed low voltage complexes with flattened *T* waves.

Dr. Hamilton's second patient suffering from Simmonds's disease was a woman, aged thirty-three years, who had complained for three years of lassitude, sensitivity to cold, loss of one stone in weight, constipation, and scanty infrequent menstruation. She had borne three children with uncomplicated deliveries. She had been treated for anæmia for three years. On examination of the patient, in the skin were seen alternating areas of light brown pigmentation and leucoderma on the face, neck and arms. There was no abnormal pigmentation of the mucosa. The head hair was lifeless, the axillary hair was scanty. The blood pressure was 120 millimetres of mercury, systolic, and 90 millimetres, diastolic, but on hot days fell to 105 millimetres of mercury, systolic, and 90 millimetres, diastolic. X-ray examination of the chest revealed a small heart shadow; the electrocardiogram showed low voltage complexes with flattened *T* waves. There was impaired excretion of a water load (Kepler-Power test), and impaired recovery from hypoglycæmia induced by insulin injected intravenously (one-third of the standard dose of 0·1 unit per kilogram of bodyweight). There were 264 eosinophile cells per cubic millimetre of blood when the patient was fasting and 240 per cubic millimetre four hours after the intramuscular injection of 25 milligrammes of ACTH. Treatment was instituted with desoxycorticosterone acetate, thyroid and testosterone, and improvement followed until suppurative mastitis developed in the left breast. That infection persisted despite antibiotics and surgical drainage for three months, when rapid resolution followed the oral administration of cortisone, 12·5 milligrammes per day.

Autopsy material from a third case of Simmonds's disease was presented. The patient was a woman, aged fifty-nine years, who had had severe post-partum hæmorrhage after delivery nineteen years prior to her admission to hospital. No lactation followed the birth, the hair of the head and the eyebrows and axillary and pubic hair began to fall out, and she complained of lassitude, intolerance of cold, and constipation. She had been under treatment for anæmia. Examination of the patient showed no loss of weight; she had a fine, slightly scaly, waxy skin, axillary and pubic hair was lost, her voice was coarse and monotonous, and her facies was drooping and immobile. The blood pressure was 220 millimetres of mercury, systolic, and 140 millimetres, diastolic. The hæmoglobin value was 83% (12·1 grammes *per centum*). There were 726 eosinophile cells per cubic

millimetre of blood when the patient was fasting, and 407 per cubic millimetre four hours after an intravenous injection of 25 milligrammes of ACTH. There was impaired excretion of a water load (Kepler-Power test) and impaired recovery from hypoglycæmia induced by insulin injected intravenously (one-third of the standard dose of 0·1 unit per kilogram of body weight). The basal metabolic rate was −30%, and the blood cholesterol content 230 milligrammes per 100 millilitres. The electrocardiogram showed low voltage complexes and flattened *T* waves. The patient had responded well to treatment with desoxycorticosterone acetate, thyroid and testosterone, but failed to return for maintenance therapy. She collapsed suddenly and died within twenty-four hours twelve months after her discharge from hospital. At autopsy, almost complete replacement of the anterior lobe of the pituitary gland by fibrous tissue was found, and extreme atrophy of the thyroid and adrenal glands.

Dr. Hamilton Smith pointed out that each of these three patients had presented first with anæmia, which in two cases was thought to be pernicious anæmia. Of the three, two showed complete lack of axillary and pubic hair, while the third showed scanty axillary hair and pigmentation of the skin, suggestive of Addison's disease. This lack of axillary and pubic hair strongly suggested the diagnosis of hypopituitarism. None of the patients had undergone severe loss of weight. In only one case was the classical history of post-partum hæmorrhage obtainable, the cause of the hypopituitarism remaining obscure in the two other cases.

Dr. Hamilton Smith emphasized the value of three investigations during consideration of the possibility of hypopituitarism: the renal response to a water load (Kepler-Power test), the electrocardiogram, and the response of the circulating eosinophile cells to ACTH injected intramuscularly. Of the three tests, he considered the most useful the renal response to a water load (Kepler-Power test). If the excretion of urine over five hours was significantly less than the dose of water taken (10 millilitres per pound of body weight), and if the deficiency in output was corrected on repetition of the test four hours after the oral administration of 100 milligrammes of cortisone, primary or pituitary adrenal insufficiency was probably present. The two surviving patients were well on maintenance therapy with thyroid, cortisone and desoxycorticosterone acetate.

Addison's Disease.

Dr. Hamilton Smith next presented a man, aged forty-nine years, who was shown to be suffering from Addison's disease with inconspicuous pigmentation of the skin and no pigmentation of the mucosa. He had been tired and weak for three months and was subject to "dizzy spells". He had felt nervous, anxious and afraid to meet people. He had lost two stone in weight in the previous twelve months, his appetite being capricious. He was subject to diarrhœa, especially when emotionally upset. Examination revealed him to be a moderately undernourished man with light brown pigmentation of the forehead and neck. His blood pressure on his admission to hospital was 120 millimetres of mercury, systolic, and 80 millimetres, diastolic. During hot weather, which precipitated a bout of diarrhœa, the blood pressure fell to 95 millimetres of mercury, systolic, and 65 millimetres, diastolic. The first abnormal finding on investigation was excessive intestinal hurry at barium meal examination. The heart shadow on X-ray examination of the chest was seen to be small. The hæmoglobin value was 75%, the plasma cell volume 50%. There were 460 eosinophile cells per cubic millimetre of blood when the patient was fasting, and 530 per cubic millimetre after 25 milligrammes of ACTH infused intravenously over eight hours on two successive days. The renal excretion of a water load (Kepler-Power test) was impaired. Urinary 17-ketosteroid excretion was 3·5 milligrammes per twenty-four hours. The Mantoux test produced a positive reaction to a dilution of 1 in 10,000. Treatment was begun with desoxycorticosterone acetate, and while the patient was under treatment an opacity developed in the second left intercostal space, which was seen on X-ray examination of the chest. Attempted culture of the contents failed to yield a growth of tubercle bacilli. The erythrocyte sedimentation rate was 18 millimetres in one hour. Cortisone by mouth, 50 milligrammes daily in divided doses, was administered in addition to desoxycorticosterone acetate, streptomycin and para-aminosalicylic acid. The radiological opacity gradually decreased in size over six months' observation. The desoxycorticosterone was administered in the form of desoxycorticosterone trimethyl acetate, in doses of 25 milligrammes and later 50 milligrammes by intramuscular injection every four weeks. The dose of desoxycorticosterone was determined largely by observation of the plasma cell volume and the size of the heart shadow on X-ray examination of the chest. Less sensitive indicators of dosage were body weight and blood pressure.

Dr. Hamilton Smith emphasized first the difficulty of distinguishing between psychoneurosis and Addison's disease in the absence of conspicuous pigmentation, and secondly, the satisfactory response of the pulmonary lesion to antibiotics during the administration of cortisone.

Diabetes and Liver Disease.

Dr. Hamilton Smith's last two patients presented the clinical picture of diabetes in the presence of liver disease. The first of these patients was a man, aged twenty-nine years, who had been an alcoholic for some years. Glycosuria (to 2%) was found on routine examination. There was no family history of diabetes. The fasting blood sugar content was 173 milligrammes per 100 millilitres, the result of the glucose tolerance test being typically diabetic. On examination of the patient, the liver was firm and enlarged to four centimetres below the right costal margin. Liver biopsy showed severe fatty changes with a moderate increase of fibrous tissue. During an intravenous glucose tolerance test, the serum inorganic phosphate level dropped by 25% of the fasting level.

The second of the two patients with diabetes and liver disease was a man, aged fifty-seven years, who had been an alcoholic for a number of years. He had been under treatment for diabetes with diet and insulin for fourteen years. There was no family history of diabetes. On his admission to hospital glycosuria (1·5%) was present, and the fasting blood sugar content was elevated (245 milligrammes per 100 millilitres). The liver was firm and enlarged three centimetres below the right costal margin. The cephalin flocculation test produced a strongly positive reaction, and liver biopsy revealed fatty infiltration and considerable deformity of the hepatic architecture. The blood reacted strongly to the Wassermann test. There was no fall of the serum inorganic phosphate level during the performance of an intravenous glucose tolerance test.

Dr. Hamilton Smith pointed out how liver disease could mimic *diabetes mellitus* as in the first of these two cases. In this case, during the intravenous administration of glucose, the serum inorganic phosphate content fell by 25% of the fasting level. In true *diabetes mellitus* an insignificant fall of the serum inorganic phosphate level was found, which reflected the impaired peripheral utilization of glucose found in that disorder. In the second case, in view of the lack of any fall of the serum inorganic phosphate level following the administration of glucose, Dr. Hamilton Smith suggested that liver disease probably coexisted with true *diabetes mellitus*.

Intracranial Hæmorrhage of Traumatic Origin.

DR. ERIC SEAL, representing Dr. Frank Morgan, presented six patients who had had unusual intracranial hæmorrhage of traumatic origin. Two of them had had extradural hæmatomata of the posterior fossa, one a delayed intracerebellar hæmorrhage, one an intradural hæmatoma, one a large intraventricular clot, and the last an extensive intracerebral hæmorrhage in the parieto-temporal region.

In the cases of extradural hæmorrhage in the posterior fossa, the histories dated back two months and two weeks respectively. Both patients presented with headache and drowsiness. Both had papilœdema and a sixth nerve palsy, and both had radiological evidence of fracture or fracture separation about the parieto-mastoid suture line.

The patient with delayed intracerebellar hæmorrhage had jolted her head slightly in a motor-car accident three weeks prior to her admission to hospital, and one week later complained of severe pain in the occipital and cervical region, and intermittent attacks of drowsiness. On examination of the patient, there had been found neck stiffness, nystagmus (especially to the left), ataxia and weakness of the left upper limb and an equivocal left plantar reflex. X-ray films of the skull and spine were of normal appearance, and no abnormality was detected in the cerebro-spinal fluid. Dr. J. Billings had diagnosed a hæmorrhage into the left cerebellar hemisphere, and this was confirmed at operation. After the evacuation of the hæmorrhage the patient made a complete recovery, and at the time of the meeting she was free of signs and symptoms.

The patient with the intradural hæmatoma had suffered from headaches with slight drowsiness for three weeks after a hit on the head with a wooden block. There were no clinical signs. Routine X-ray examination of the skull

revealed a fracture in the left temporal region, and air encephalography showed displacement of the ventricular system to the right. At operation a large clotted hæmorrhage was removed from between the layers of the dura. At the time of the meeting the man was well.

The patient with the intraventricular hæmatoma had become deeply comatose some twelve hours after falling over while drunk. He was aged forty-three years. The tone in his right limbs was increased, and the right plantar reflex, and later the left, became extensor in type. The cerebro-spinal fluid was evenly blood-stained. Ventriculography revealed the presence of a very large clot in the anterior half of the left lateral ventricle bulging into the right lateral and third ventricles. It was removed through a left-sided transcortical incision. The patient responded well physically, but had later to be certified insane and admitted to a mental hospital.

The sixth patient had struck the back of her head when she fell backwards tugging at a root in her garden, two days previous to her admission to hospital. She had been found to be aphasic three hours later, with right hemiparesis. The cerebro-spinal fluid was blood-stained, and a linear fracture was seen straddling the mid-line in the parieto-occipital area. Ventriculography disclosed a large shift of the ventricles to the right. The brain needle entered the hæmorrhagic cavity, which was outlined with air after dark fluid blood had been aspirated. Later, transcortical removal of large clots in the left parieto-temporal region was carried out. Improvement had been slow but encouraging. She was now able to walk, and had recovered some use of speech with the help of the speech therapist, though most of her utterances consisted of jargon. The right arm was still weak and the hand paralysed. Her age was sixty years.

Peutz's Syndrome: Facial Pigmentation with Intestinal Polyposis.

Dr. Luke Murphy first showed a schoolgirl, aged seventeen years, who gave a history of nocturnal vomiting attacks of ten months' duration. These attacks occurred initially every few weeks, the patient being awakened by a sense of nausea closely followed by vomiting. The vomitus contained remnants of the evening meal, occasionally food taken at lunch time. In the later weeks the attacks had become more frequent, occurring both night and day, and being accompanied by anorexia and loss of weight. There was no pain whatsoever or any other symptoms.

Initial physical examination showed the patient to be a tall, thin girl with a fair complexion. The pulse, temperature and blood pressure were normal. The tongue was moist and clean. Around the mouth were collected pigmented spots resembling freckles, but somewhat darker. These extended to the lips, while a few were found on the nose and upper eyelids. None were found on the hands or feet. A complete physical examination, including rectal examination, revealed no other abnormality. The hæmoglobin value was 13 grammes per 100 millilitres. A barium meal X-ray examination showed a considerable six-hour residue in the stomach. The duodenum was much dilated, while the first portion of the jejunum was enormously distended, the barium passing through it slowly and producing a feathery pattern. The remainder of the bowel appeared normal. During the X-ray procedure, vomiting continued with the development of alkalotic tetany. This was corrected by intravenous therapy and immediate laparotomy was performed.

An intussusception of the first part of the jejunum due to a pedunculated polypus was found. Three other polypi were discovered in the jejunum; but the remainder of the gastro-intestinal tract was found to be free of them. All were removed and submitted to histological examination, which showed them to be adenomatous in nature. One showed carcinomatous change in its base. Convalescence was uneventful, and the patient, twelve months after operation, was free from symptoms and in good health.

Dr. Murphy said that intussusception of the small bowel in a patient in the teens was the characteristic presentation of Peutz's syndrome, associated with the characteristic facial pigmentation. The fair complexion in the present case was unusual. A dark complexion was generally regarded as a feature of the syndrome. The familial tendency found in about half the recorded cases was absent in this instance.

Radiological and Gastroscopic Demonstration.

The remainder of the demonstration consisted of a comparison of radiological and gastroscopic appearances of a number of lesions examined during the previous twelve months. The gastroscopic appearances were represented by coloured drawings made during the examinations.

Several examples of benign gastric ulcer and gastric carcinoma were shown. In three cases of lesser curvature ulcer seen radiologically, the clear-cut regular edge and smooth pale base of the benign ulcer were visible. These contrasted sharply with the appearance of a carcinomatous prepyloric ulcer, which had an irregular serpiginous nodular edge with bleeding spots and a dirty grey base.

A woman, aged fifty-eight years, presenting with hæmatemesis, was shown radiologically to have a small regular oval filling defect on the posterior wall of the stomach near the *incisura angularis*. The gastroscopic picture showed a smooth slug-like benign gastric polypus which was later resected.

A woman, aged sixty years, with a ten years' history of epigastric pain after meals, was found radiologically to have a diverticulum of the stomach high on the posterior wall. The gastroscopic pictures showed the large crater lined by normal mucosa. A peristaltic wave was seen to lift the floor, indicating the presence of muscle in the wall of the diverticulum.

A man, aged fifty-two years, had had a gastro-enterostomy established three years previously for duodenal ulceration. Recently he had suffered bouts of epigastric pain and vomiting after meals. The radiologist reported a filling defect on the lower margin of the gastro-enterostomy stoma. The gastroscopic picture showed this to be due to a large fold of redundant gastric mucosa protruding into the lumen of the stomach. The stoma and the gastric and jejunal mucosa were seen to be otherwise normal.

Myasthenia Gravis.

Dr. John Billings presented two patients suffering from *myasthenia gravis*. The first was a woman, aged fifty-four years, who some eighteen years previously had noticed intermittent drooping of the left eyelid for some months. This was frequently accompanied by diplopia. The disorder had spontaneously recovered, only to recur some eleven months prior to the present examination. On examination of the patient, drooping of the left eyelid and weakness of the lateral rectus muscle and of the superior rectus muscle of the left eye were present. A trial dose of "Prostigmin" had produced dramatic improvement, and the patient was now being satisfactorily maintained on eight tablets of "Prostigmin" daily. X-ray examination of the chest revealed no abnormality. Dr. Billings remarked that these examples of mild myasthenia were not uncommon. The medical treatment was very satisfactory, and there was a pronounced tendency to spontaneous remission.

The second patient, a man, aged fifty-three years, presented a much more difficult problem in treatment. Over a period of some six months he had developed progressive bilateral ptosis with almost constant diplopia. On examination of the patient, the severe ptosis was evident, and there was almost complete immobility of the eyeballs. No muscular weakness was evident elsewhere. An injection of 2·5 milligrammes of "Prostigmin", preceded by 1/150 grain of atropine, produced complete relief of the ptosis and very considerable improvement in the eye movements; but the improvement had been accompanied by widespread fasciculation of the facial movements, numbness of the lips and tongue and a movement of the bowels. It had been necessary to administer atropine by mouth as well as "Prostigmin" tablets in order to produce any reasonable relief of the myasthenic symptoms. Dr. Billings said that ordinarily atropine was better avoided in the treatment of myasthenia, because by masking the side effects of over-dosage of "Prostigmin" it could lead to over-treatment. In large amounts "Prostigmin" had a curarizing effect. The patient was also receiving small doses of ephedrine and X-ray therapy to the thymic region. X-ray examination of the chest had revealed no abnormality.

Subarachnoid Hæmorrhage.

Dr. Billings also showed some patients who had suffered from subarachnoid hæmorrhage. In one of these cases the disorder had been regarded as a complication of meningovascular syphilis, but in others the underlying condition was an intracranial aneurysm.

Aneurysms.

Dr. Billings finally showed a number of angiograms illustrating aneurysms in different situations and discussed the improvement in the surgical teatment of this very serious disorder.

VICTORIAN BRANCH NEWS.

Supplement to Catalogue of the Library of the Medical Society of Victoria.

Journals.

Since publication of the second edition of the catalogue of the library of the Medical Society of Victoria in 1949, the following journals have been added to the library: *Acta Radiologica* Supplements commencing at No. 85, *Annals of the Rheumatic Diseases, Australasian Annals of Medicine, Carcinoma Bulletin, Family Doctor, Geriatrics, Journal of Gerontology, New York State Journal of Medicine, Obstetrics and Gynecology, Postgraduate Medical Journal.*

Through the generosity of certain members, virtually complete current files of the following journals are now held: *American Practitioner, The British Journal of Dermatology and Syphilology, Journal-Lancet, Proceedings of the Mayo Clinic, Minnesota Medicine, New Orleans Medical and Surgical Journal, Pennsylvania Medical Journal, United States Armed Forces Medical Journal.*

Year Books.

The library subcommittee also wishes to inform members that the Year Books of all the various specialties are available.

New Books.

The following is a list of books recently added to the library up to August, 1954.

Allergy.—Hansel, F. K., "Clinical Allergy", 1953.

Anæsthetics.—Collins, V. J., "Principles and Practice of Anesthesiology", 1952; Cullen, S. C., "Anæsthesia in General Practice", Third Edition, 1952; Hewer, C. L., "Recent Advances in Anæsthesia and Analgesia", Seventh Edition, 1953; Lee, J. A., "Synopsis of Anæsthesia", 1950; Mackintosh and Bannister, "Essentials of General Anæsthesia", Fifth Edition, 1952; Mushin, W., and Rendell-Baker, L., "The Principles of Thoracic Anæsthesia, Past and Present", 1953; Watson, John, "How to Give Open Ether", 1952.

Anatomy, Histology and Embryology.—Last, R. J., "Aids to Anatomy", Eleventh Edition, 1951; Ragan, C. (editor), "Connective Tissues" (Macy Foundation Conference), 1951; Wright, M. K., "Fibre Systems of the Brain and Spinal Cord", 1952.

Bones, Joints and Muscles.—Lewin, P., "Arthritis and the Rheumatic Diseases", 1951.

Chemistry, Physics and Biochemistry.—Browning, E., "Toxicity of Industrial Organic Solvents", 1953; Clark, W. M., "Topics in Physical Chemistry: A Supplementary Text for Students of Medicine", 1952.

Circulatory System.—Barron and Schott, "Cardiographic Technique, a Manual for Cardiological Technicians", 1952; Budtz-Olsen, O. E., "Clot Retraction", 1951; Custer, R. P., "An Atlas of the Blood and Bone Marrow", 1949; Evans, Wm., "Cardioscopy", 1952; Foote, R. Rowden, "Varicose Veins", 1952; International Society of Hæmatology, "Proceedings of the Third International Congress", 1950; Lepeschkin, E., "Modern Electrocardiography; Volume I, The P-Q-R-S-T-U Complex", 1951; Lipman, B. S., "Clinical Unipolar Electrocardiography", 1953; Moeschlin, S., "Spleen Puncture" (trans. by A. Piney), 1951; White, Paul, "Heart Disease", Fourth Edition, 1951; Wood, Paul, "Diseases of the Heart and Circulation", 1952.

Dermatology.—MacKenna, R. M. B., "Diseases of the Skin", Fifth Edition, 1952; Mitchell-Heggs, G. B. (editor), "Modern Practice in Dermatology", 1950; New York Skin and Cancer Unit (F. Pascher, editor), "Dermatologic Formulary", 1953; Percival, Drennan and Dodds, "Atlas of Histopathology of the Skin", 1947; Semon and Moritz, "An Atlas of the Commoner Skin Diseases", Fourth Edition, 1953; Tobias, N., "Essentials of Dermatology", Fourth Edition, 1952.

Digestive System.—Benedict, E. B., "Endoscopy as Related to Diseases of the Bronchus, Esophagus, Stomach and Peritoneal Cavity", 1951; Jones, F. Avery (editor), "Modern Trends in Gastroenterology", 1952; Lee, Alan E.: "An Introduction to Abdominal Diagnosis", 1951; Sandweiss, D. J. (editor), "Peptic Ulcer, Clinical Aspects, Diagnosis, Management", 1951.

Diseases and Care of Children.—Alm, I., "The Long-Term Prognosis for Prematurely Born Children", 1953; Brown, Vera S., and Campbell, Kate, "A Guide to the Care of the Young Child (Infant and Pre-School Ages)", Third Edition, 1951; Garrod, Batten and Thursfield, "Diseases of Children", Fifth Edition, 1953 (edited by Moncrieff and Evans), two volumes; Levine, S. Z. (editor), "Advances in Pediatrics', Volume V, 1952, Volume VI, 1953; Parmelee, A. H., "Management of the Newborn", 1952; Wilkins, E. H., "The Medical Inspection of School Children", 1952.

Endocrinology.—Selye, H., "The Story of the Adaptation Syndrome" (told in form of informal illustrated lectures), 1952.

Genito-Urinary System.—Narath, P. A., "Renal Pelvis and Ureter", 1951; Smith, Homer W., "The Kidney: Structure and Function in Health and Disease", 1951.

Hygiene and Social Medicine.—Banks, A. L., "Social Aspects of Disease", 1953; Wilkins, E. H., "The Medical Inspection of School Children", 1952.

Industrial Medicine and Workers' Compensation.—Browning, E., "Toxicity of Industrial Organic Solvents", 1953; Davis, T. A. L., "The Practice of Industrial Medicine", 1948; U.S. Public Health Service, "Health of Ferrous Foundrymen in Illinois", 1950, "Silicosis and Lead Poisoning Among Pottery Workers", 1939.

Infectious Diseases.—Burnet, Sir Macfarlane, "The Natural History of Infectious Disease", Second Edition, 1953; Levander-Lindgren, M., "Electrocardiographic Studies in Scarlet Fever (An Investigation with Special Reference to the Effect of Penicillin Treatment)", 1952; Meenan, P. N., "The Essentials of Virus Diseases", 1951; Muir, E., "Manual of Leprosy" (Supplement to First Edition), 1952; Rogers and Muir, "Leprosy" (Addendum to Third Edition), 1952.

Materia Medica and Pharmacology.—"British Pharmacopœia", 1953; Derbes and Weiss, "Untoward Reactions of Cortisone and ACTH", 1951; Ghosh, B. N., "Pharmacology, Materia Medica and Therapeutics", Nineteenth Edition, 1952; "Iodine Pharmaceuticals: A Checklist of Iodine Compounds and Preparations Used in Human Medicine", 1952; Martindale, "The Extra Pharmacopœia", Twenty-Third Edition, Volume I, 1952.

Medical Jurisprudence and Toxicology.—"The Law in Relation to the Illegitimate Child: Report of the Joint Committee on Psychiatry and the Law, Appointed by the B.M.A., London, and the Magistrates' Association", 1952; Lucas, G. H., "The Symptoms and Treatment of Acute Poisoning", 1953.

Medicine.—Aero Medical Association (United States of America), "Aviation Toxicology: An Introduction to the Subject and a Handbook of Data", 1953; "Any Questions? A Selection of Questions and Answers Published in the British Medical Journal", 1952; Beaumont and Dodds, "Recent Advances in Medicine", Thirteenth Edition, 1952; Beaumont, G. E., "Medicine: Essentials for Practitioners and Students", Sixth Edition, 1953; Birch, C. A., "Emergencies in Medical Practice", 1952; Chamberlain, E. N., "A Textbook of Medicine", 1951, "Symptoms and Signs in Clinical Medicine: An Introduction to Medical Diagnosis", Fifth Edition, 1952; Chatton, Margen and Brainerd, "Handbook of Medical Management", 1949; Conn, H. F. (editor), "Current Therapy", 1953; Dock and Snapper (editors), "Advances in Internal Medicine", Volume V, 1953; Dunlop, D. M., *et alii*, "Textbook of Medical Treatment", Sixth Edition, 1953; "Edinburgh Post-Graduate Lectures in Medicine", by various authors, 1952; Horder, Lord, and Gow, A. E., "The Essentials of Medical Diagnosis", Second Edition, 1952; Mainland, D., "Elementary Medical Statistics: The Principles of Quantitive Medicine", 1952; "Recent Advances in Chemotherapy", Volume III (Antibiotics), 1954; "Refresher Course for General Practitioners: Specially Commissioned Articles from the British Medical Journal", 1950; Walker, H., "Physical Diagnosis", 1952.

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Treatise on Scurvy: A Bicentenary Volume Containing a Reprint of the First Edition of A Treatise of the Scurvy by James Lind, with Additional Notes", 1953.

Military Medicine and Surgery.—"History of the Second World War (U.K. Medical Series): Medicine and Pathology" (edited by V. Z. Cope), 1952; "The Emergency Medical Services. Volume I: England and Wales" (by C. L. Dunn), 1952; "The Army Medical Services. Volume I: Administration" (by F. A. E. Crew), 1953; "Surgery" (edited by Zachary Cope); Walker, Alan S., "Australia in the War of 1939-1945": Volume I, "Clinical Problems of War", 1952, Volume II, "Middle East and Far East", 1953.

Miscellaneous.—Auerbach, C., "Notes for Introductory Courses in Genetics", Third Edition, 1951; Bett, W. R., "The Preparation and Writing of Medical Papers for Publication"; "The Collected Papers of the Commonwealth Serum Laboratories", Volume I, 1920-1944, Volume II, 1945-1953; Commonwealth Department of Health, "Pesticides: A Review of Their Uses, Properties and Hazards", 1951; Stone, J. E., "Hospital Organization and Management", Fourth Edition, 1952.

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Acta Radiologica Supplements.—90, *"Lesions nouvelles du poignet"* (Bugnion), 1951; 91, "Uterine Cervix and Isthmus Under Normal and Pathological Conditions (a Clinical and Roentgenological Study)" (Asplund), 1952; 92, "Carcinoma of the Uterine Cervix, Incidence and Influence of Age (Lindell), 1952; 93, "Contributions to the Theory and Technique of Quantitative Autoradiography with P^{32} with Special Reference to the Granulosa Tissue of the Graafian Follicles in the Rabbit (Odeblad)", 1952; 94, "Mass, Lipids, Pentose Nucleoproteins and Proteins Determined in Nerve Cells by X-Ray Microradiography" (Brattgard and Hyden), 1952; 95, "Urethrography in the Male with a Highly Viscous, Water-Soluble Contrast Medium, Umbradil-Viscous U", 1952; 96, "The Importance of Adequate Stimulation for the Chemical Composition of Retinal Ganglion Cells during Early Post-Natal Development", 1952; 97, "A Roentgenologic Study of the Coronary Arteries in the Living" (Guglielmo and Guttadauro); 98, "Radiotherapy in Cancer of the Breast, with Particular Reference to the Value of Pre-Operative Irradiation as a Supplement to Radical Mastectomy (Analysis of 1418 New Cases)" (Kaae), 1952; 99, Investigations on Backflow in Retrograde Pyelography (a Roentgenological and Clinical Study)" (Kohler), 1953; 100, "Post-Irradiative Prophylactic Extraperitoneal Lymphadenectomy in Carcinoma of Uterine Cervix" (Gorton), 1953; 101, "Value of Tomography in Examination of the Intrapulmonary Bronchi" (Lodin), 1953; 102, "Microangiography" (Bellman), 1953; 103, "Principles of Vertebral Tomography" (Bokstrom), 1953; 104, "Meningiomas of the Posterior Fossa" (Castellano and Ruggiero), 1953. (For Nos. 85-89 see previous supplement to the catalogue.)

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

VACCINATION.[1]

[From *The Australian*, August 20, 1840.]

THE committee of the Sydney Dispensary beg to apprise the public that children will be vaccinated by their medical attendant at their rooms, Pitt Street, near Market Street, on Thursday the sixth instant and every succeeding Thursday between the hours of two and three p.m. One shilling will be charged to be returned on presenting the child on the eighth day for inspection.

Vaccine virus will be furnished to medical practitioners in the interior by Dr. McKellar, medical attendant, through the post office, free of expense.

Resolution passed at the last meeting of the committee "That the clergymen of Sydney be respectfully requested to intimate from the pulpit to their congregations, the propriety of parents immediately vaccinating their children, and that the same will be done gratuitously on each Thursday at the above hour".

W. HUTCHINSON, Chairman.
J. MCGARVIE, Honorary Secretary.

Correspondence.

A CORRECTION.

SIR: I would like to draw your attention to some errors occurring in the report of the seventh annual meeting of the Urological Society of Australasia in the journal of February 12.

On page 249 it is suggested that I thought cortisone and DOCA might "reduce hypertension" in patients with anuria. I intended to convey the impression that these substances might assist in alleviating *hypotension* induced by shock, which is often a major factor in the production of anuria. My remarks were in answer to a question raised by Dr. R. J. Walsh in his discussion.

In the subsequent paragraph "ion" should be read for "iron" in the fourth last line.

Yours, etc.,

Clinical Research Unit, Royal Prince Alfred Hospital, Sydney.
February 15, 1955.

C. R. B. BLACKBURN.

MONGOLISM.

SIR: A number of parents of mongol children, in this State at any rate, are being told by doctors to have such a child mentally certified as soon as possible after birth and to let it be generally known that the child has died.

What the legal position might be in regard to giving such advice I do not know, but surely the attitude of the community towards mental deficiency is becoming sufficiently enlightened to make such subterfuge out of date; and surely we, as doctors, should be the last to encourage it. The community, today, is coming to realize there is no stigma attached to having a spastic or a mongol child and that such children are no longer to be regarded as skeletons in cupboards. Spastic and subnormal children's centres and committees are doing splendid work for these children and their parents, and doctors, generally, must take a more realistic view.

When I diagnose a child patient as a mongol, I explain as kindly as I can to the parents what the child's future is likely to be and suggest, particularly if it is a city child, that it be voluntarily certified and sent to the children's ward of a mental hospital. Here the child is happy, as it has its bodily wants attended to and, to a limited extent, receives some mental training. But I encourage the parents to visit the children at intervals, and I see no earthly reason why they cannot tell neighbours and relatives in a matter-of-fact way that the child is a mongol and is in hospital.

With others I look forward to the day when governments will make provision for special wards in mental hospitals for such special categories of mental patients as mongols, certain spastics and old people with moderate degrees of cerebral degeneration.

Yours, etc.,

Ipswich, Queensland,
February 17, 1955.

H. S. PATTERSON.

[1] From the original in the Mitchell Library, Sydney.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

General Revision Course.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that the annual general revision course will be held for two weeks from Monday, May 16, to Friday, May 27, 1955, inclusive, under the supervision of Dr. J. W. Graham. The programme is as follows:

Monday, May 16: 9.15 a.m., registration; 9.45 a.m., opening of course by Dr. A. M. McIntosh; 10 a.m., election of course chairman followed by a review of the course by the honorary director; 10.45 a.m., "Seminar on Cardio-Vascular Disease—Hypertension": "Types and Clinical Varieties", Dr. T. M. Greenaway, "Pathology", Dr. V. J. McGovern, "Treatment, with Special Reference to Hypotensive Drugs", Dr. J. K. Maddox, "Management of Cerebral Complications", Dr. K. B. Noad; 2.15 p.m., "Seminar on Cardio-Vascular Disease—Coronary Artery Disease", Dr. F. L. Ritchie; 4.15 p.m., "The Arrhythmias", Dr. Justin Markell.

Tuesday, May 17: 9.15 a.m., "Seminar on Cardio-Vascular Disease—Management of Congestive Heart Failure and Chronic Heart Disease", Dr. Hales Wilson; 10.45 a.m., "Surgical Treatment of Heart Disease": "Selection of Patients", Dr. Sinclair Smith, "Special Methods of Investigation", Dr. E. J. Halliday, "Surgical Aspects", Dr. F. H. Mills, "Results of Treatment", Dr. John H. Halliday; 2.15 p.m., "Seminar on Cardio-Vascular Disease—Peripheral Vascular Disease": "Medical Aspects", Dr. A. E. McGuinness, "Surgical Aspects", Dr. John Loewenthal; 4 p.m., "Heart Disease and Pregnancy", Dr. John Cobley, "Hypertension and Pregnancy", Dr. Robert Back.

Wednesday, May 18: 9.15 a.m., "Seminar on Cardio-Vascular Disease—Rheumatic Carditis", Dr. Lawrence Hughes; 10.45 a.m., "Industrial Aspects of Heart Disease", Dr. F. A. E. Lawes; 11.45 a.m., "The Significance of Rectal Bleeding"; 2.15 p.m., "Seminar on Cancer—Early Diagnosis": "Alimentary Tract", Dr. R. M. Rawle, "Respiratory Tract", Dr. H. Maynard Rennie, "Female Genital Organs", Dr. R. H. Macdonald, "General Comments on Exfoliative Cytology", Dr. E. Hirst; 8.15 p.m., in the Great Hall of the University of Sydney, Eighth Annual Post-Graduate Oration, "The Life and Times of George Bennett" (orator, Dr. V. M. Coppleson).

Thursday, May 19: 9.15 a.m., "Electrocardiographic Conference", Dr. W. A. Seldon; 10.45 a.m., "X-Ray Conference", Dr. Mary Cronin, Dr. V. J. Kinsella and Dr. Bruce Hall; 11.45 a.m., "Seminar on Anæsthesia": chairman, Dr. L. T. Shea, speakers, Dr. A. Distin Morgan, Dr. C. N. Paton and Dr. P. L. Jobson; 2.15 p.m., "Seminar on Surgery—Diseases of the Breast", Dr. Kathleen Cuningham.

Friday, May 20: "Seminar on New Drugs and Therapeutic Measures": chairman, Sir Archibald Collins, speakers, Dr. A. W. Morrow, Dr. Kenneth Hughes, Dr. Keith Harrison and Dr. Alan Sharp.

Monday, May 23: 9.15 a.m., at the Royal Alexandra Hospital for Children, "Symposium—Chronic and Recurrent Cough during Childhood": "Differential Diagnosis", Dr. D. G. Hamilton, "Management of Chronic and Recurrent Infections of the Respiratory Tract", Dr. John Alexander, "Management of Allergic Disorders of the Respiratory Tract", Dr. S. E. L. Stening, "The General Practitioner's Approach to Congenital Heart Disease", Dr. Douglas Stuckey; 2 p.m., at the Royal Alexandra Hospital for Children, "Clinical Demonstration of Pædiatric Problems (Medical)", Professor Lorimer Dods, "Clinical Demonstration

of Pædiatric Problems (Surgical)", Dr. T. Y. Nelson; 8 p.m., at the Stawell Hall, Third Conference on Medical Education, "Post-Graduate Education of the Practising Doctor".

Tuesday, May 24: 9.15 a.m., "Seminar on Gynæcological Problems in General Practice": chairman, Dr. G. G. L. Stening, "Sterility", Dr. R. B. C. Stevenson, "Uterine Cancer", Malcolm Stening, "Ovarian Cancer", Dr. H. K. Porter, "Endometriosis", Dr. A. R. H. Duggan; 2.15 p.m., "Seminar on Obstetrical Problems in General Practice": Chairman, Professor Bruce T. Mayes, "Breech Delivery", Dr. W. Cunningham, "'Pitocin' in Induction of Labour", Dr. J. Chesterman, "Toxæmia of Pregnancy", Dr. Ida Saunders, "Ante-Partum Hæmorrhage", Dr. Angus Murray; 8.15 p.m., at the Stawell Hall, "Medical Problems of Atomic Warfare", Major-General F. Kingsley Norris, C.B., C.B.E., D.S.O.

Wednesday, May 25: 9.15 a.m., "Legal Pitfalls in General Practice", Dr. W. Brooks; 10.45 a.m., "Fluid and Electrolyte Balance", Dr. C. R. B. Blackburn; 11.45 a.m., "Hydrocortisone in Joint Disease", Dr. Selwyn Nelson; 2.15 p.m., "Seminar on Trauma": "Traumatic Surgery of Hand", Dr. P. H. Greenwell, "Chest Injuries", Dr. H. M. Windsor, "Abdominal Injuries", Dr. Norman Wyndham.

Thursday, May 26: 9.15 a.m., at the Northcott Neurological Centre, "Neurological Problems in General Practice": "Disseminated Sclerosis", Dr. Eric Susman, demonstration of three interesting neurological cases, "The Management of Acute Head Injuries in General Practice", Dr. S. M. Morson; 2.15 p.m., at Broughton Hall Psychiatric Clinic, demonstration of "Psychiatric Problems in General Practice" by the staff.

Friday, May 27: 9.15 a.m., "Common Skin Conditions", Dr. Adrian Johnson; 10.45 a.m., "Seminar on Various Diagnostic Procedures": "Ear, Nose and Throat", Dr. Volney Bulteau, "Ophthalmology", Dr. Conrad Blakemore, "Gastroscopy and Sigmoidoscopy", Dr. S. Goulston; 2.15 p.m., "Question Time": chairman, the honorary director, Dr. V. M. Coppleson, panel, Dr. Innes Brodziak, Dr. Douglas Miller, Dr. Bruce Williams and Dr. Angus Murray.

All lectures will be held in the Stawell Hall, 145 Macquarie Street, Sydney, unless otherwise indicated.

Fee for attendance will be £12 12s. full time, or £6 6s. for one week or part time. Early written application is essential and should be made to the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497, BU 4498. Telegraphic address: "Postgrad, Sydney."

Fees, fares and expenses for this course are allowable deductions for taxation under "Taxation—File No. AF/1865".

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

Programme for April, 1955.

Higher Qualifications.

CLASSES for candidates for Part I of higher qualifications which commenced at the University in February and early March will be continued in April.

Surgery.

The course in surgery at the Royal Melbourne Hospital will continue from March 21 to April 29.

Ophthalmology.

A course in ophthalmology, including special pathology, will be conducted by the Victorian Section of the Ophthalmological Society of Australia (British Medical Association), commencing on April 4. Lectures and demonstrations will be held about four afternoons each week until September, from 5 till 6 p.m. The fee for this course is £31 10s., and enrolments should be made with the Post-Graduate Committee at 394 Albert Street, East Melbourne.

Country Courses.

On April 2 a week-end refresher course will be held at Yallourn. The speakers and their subjects will be as follows: Dr. Keith Bradley, "Operations in the Autonomic Nervous System"; Dr. J. L. Frew, "The Management of Hypertension"; Dr. V. L. Collins, "Deficiency Disease of Childhood".

On April 13, at 2.30 p.m., a demonstration will be held at Flinders Naval Depot. Dr. Ian Wood will speak on "Diagnosis and Treatment of Chronic Epigastric Pain". This is by arrangement with the Royal Australian Navy.

Courses Commencing in May and June.

Medicine for M.D., Part II, and M.R.A.C.P.

Commencing on June 6, the honorary medical staff of Saint Vincent's Hospital, Melbourne, will conduct a course in medicine on six mornings a week for eight weeks. This will be suitable for candidates for M.D., Part II, and M.R.A.C.P. There will be ward rounds, clinical demonstrations, lectures, case presentations and demonstrations of X-ray films, electrocardiograms, pathology specimens *et cetera*. Included in the course will be two sessions at the Infectious Diseases Hospital, Fairfield.

The fee for the course will be £31 10s., and enrolments should be made through the Melbourne Medical Post-Graduate Committee, 394 Albert Street, East Melbourne, who will supply further information on request. If preferred, inquiries may be sent direct to the dean of the clinical school at the hospital. A detailed programme will be available shortly.

Psychiatry for D.P.M.

A course of lectures in psychiatry suitable for candidates for the diploma in psychological medicine has been arranged by the Australasian Association of Psychiatrists, Victorian Branch, for the Melbourne Medical Post-Graduate Committee. These will commence in early June and will be held in June, July and August in Melbourne at 45 Spring Street, on Monday and Thursday evenings from 8.15 p.m. There will also be some Saturday sessions occupying the afternoon and evening. Details of subjects and speakers and the commencement date will be announced soon. Candidates wishing to make further inquiries are invited to get into touch with the Post-Graduate Committee, 394 Albert Street, East Melbourne.

This course is distinct from that provided by the Victorian Department of Mental Hygiene, announced to British Medical Association members in the Post-Graduate Committee's February and March programmes and in THE MEDICAL JOURNAL OF AUSTRALIA, under the title "Psychiatry for D.P.M." and "Basic Post-Graduate Psychiatry". The Department's course is designed as basic study in post-graduate psychiatry, which is suitable for medical officers training for the D.P.M.

Other facilities for post-graduate seminars and demonstrations will be available during June, July and August, provided by the psychiatric departments of public hospitals, by the Institute for Psychoanalysis, and by individual members of the Victorian Branch of the Australasian Association of Psychiatrists. Those wishing to make further inquiries regarding these other facilities are invited to write to Dr. Ainslie Meares, Honorary Secretary, Victorian Branch, The Australasian Association of Psychiatrists, 45 Spring Street, Melbourne.

Course for D.L.O., Part II.

A course of approximately 60 lectures and demonstrations has been drawn up; should the demand be sufficient, this will be conducted before the Part II examination for the diploma of laryngology and otology next October. It will be necessary for the course to commence about May, and candidates should inquire immediately from the Melbourne Medical Post-Graduate Committee, 394 Albert Street, East Melbourne.

Courses for Part II D.D.R. and M.C.R.A. in Diagnostic Radiology.

Commencing in May, the Post-Graduate Committee, in consultation with the College of Radiologists of Australasia, will conduct a course in radiodiagnosis, probably on two late afternoons a week. In conjunction with this course, lectures will be arranged in special pathology on one afternoon a week in June and July. Inquiries should be addressed to the Post-Graduate Committee.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE following notes relate to the Australasian Medical Congress (British Medical Association), Ninth Session, which is to be held at the University of Sydney from August 20 to 27, 1955.

Office-Bearers of Sections.

The presidents and honorary secretaries of the scientific sections are as follows:

Section of Anæsthesia.

President: Dr. H. J. Daly (New South Wales).

Honorary Secretary: Dr. P. L. Jobson (New South Wales).

Section of Dermatology.

President: Dr. W. C. T. Upton (South Australia).

Honorary Secretary: Dr. M. Havyatt (New South Wales).

Section of History of Medicine.

President: Dr. C. Craig (Tasmania).

Honorary Secretary: Dr. A. M. McIntosh (New South Wales).

Section of Medicine and Experimental Medicine.

President: Dr. W. W. S. Johnston (Victoria).

Honorary Secretary: Dr. K. S. Harrison (New South Wales).

Section of Naval, Military and Air Force Medicine and Surgery.

President: Air Vice-Marshal E. A. Daley (Victoria).

Honorary Secretary: Dr. J. F. C. Cobley (New South Wales).

Section of Neurology and Psychiatry.

President: Dr. A. J. M. Sinclair (Victoria).

Honorary Secretary: Dr. I. G. Simpson (New South Wales).

Section of Obstetrics and Gynæcology.

President: Dr. G. Simpson (Victoria).

Honorary Secretary: Dr. F. A. Bellingham (New South Wales).

Section of Ophthalmology.

President: Dr. A. E. F. Chaffer (New South Wales).

Honorary Secretary: Dr. E. J. Donaldson (New South Wales).

Section of Orthopædics.

President: Dr. E. F. West (South Australia).

Honorary Secretary: Dr. A. I. Rhydderch (New South Wales).

Section of Oto-Rhino-Laryngology.

President: Dr. A. Kenneth Green (Queensland).

Honorary Secretary: Dr. T. H. O'Donnell (New South Wales).

Section of Pathology, Bacteriology, Biochemistry and Forensic Medicine.

President: Professor F. R. Magarey (New South Wales).

Honorary Secretary: Dr. A. E. Gatenby (New South Wales).

Section of Pædiatrics.

President: Dr. Kate I. Campbell (Victoria).

Honorary Secretary: Dr. S. E. J. Robertson (New South Wales).

Section of Public Health and Industrial Medicine.

President: Dr. H. M. L. Murray (Tasmania).

Honorary Secretary: Dr. R. T. C. Hughes (New South Wales).

Section of Radiology and Radiotherapy.

President: Dr. B. L. W. Clarke (Queensland).

Honorary Secretary: Dr. D. B. Wightman (New South Wales).

Section of Rehabilitation and Physical Medicine.

President: Dr. C. W. Anderson (Western Australia).

Honorary Secretary: Dr. B. G. Wade (New South Wales).

Section of Surgery.

President: Dr. Alan H. Lendon (South Australia).

Honorary Secretary: Dr. A. C. R. Sharp (New South Wales).

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED FEBRUARY 19, 1955.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.[2]	Australia.[3]
Acute Rheumatism	6(8)	2(1)	3	..	2(2)	..	..	..	13
Amœbiasis	..	..	1	..	..	..	..	..	1
Ancylostomiasis	..	..	..	..	1	..	..	..	1
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	3	..	..	..	..	..	..	..	3
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	4	19(6)	..	1	7(6)	..	..	..	31
Diphtheria	3(1)	2(1)	2(1)	..	9(8)	..	..	..	16
Dysentery (Bacillary)	..	8(8)	2(1)	..	7(3)	..	..	..	17
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1	..	..	..	..	..	..	1
Infective Hepatitis	64(35)	66(24)	..	6	1	1	..	..	138
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	4(1)	..	..	..	..	..	4
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	1	..	2(2)	..	..	..	..	..	3
Ophthalmia	..	..	..	..	1	..	..	..	1
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	13(5)	14(6)	14(3)	2(2)	..	..	..	..	43
Puerperal Fever	..	..	2	..	..	..	..	..	2
Rubella	..	38(21)	..	2	6(5)	..	..	..	46
Salmonella Infection	..	..	..	..	5	..	..	..	5
Scarlet Fever	7(1)	13(11)	6(2)	1	1(1)	..	..	..	28
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	2	..	..	..	..	..	2
Trachoma	..	..	..	..	1	..	..	..	1
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	43(30)	38(27)	27(19)	5(3)	17(11)	3(2)	..	..	133
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	1	..	..	..	2
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

[2] Figures not available.

[3] Figures incomplete owing to absence of returns from Northern Territory and Australian Capital Territory.

Section of Tropical Medicine.

President: Professor A. H. Baldwin (New South Wales).

Honorary Secretary: Dr. C. J. N. Leleu (New South Wales).

Scientific Papers.

Members and intending members of Congress who desire to offer papers for presentation at meetings of sections are reminded that offers of papers will be received by honorary secretaries of sections until March 31, 1955. Authors must indicate at least the title of the proposed paper with a précis of its subject if the completed paper is not submitted.

All correspondence to honorary secretaries of sections should be addressed to the Congress office, 135 Macquarie Street, Sydney.

Obituary.

JAMES HARDIE NEIL.

MR. J. H. NEIL, who died in Auckland on January 28, was distinguished in his field of ear, nose and throat surgery and a well-known public figure in New Zealand.

He was born in 1875 and studied medicine at the University of Otago. He practised in Auckland for many years, being ear, nose and throat surgeon to Auckland Hospital and the Mater Misericordiæ Hospital. His textbook on ear, nose and throat nursing reached its fourth edition in 1948. He was an early master of bronchoscopy, and later, about 1935-1936, carried out a painstaking series of dissections of human and animal lungs, and wrote on bronchial anatomy.

In the South African War he was surgeon captain to the Fourth New Zealand Mounted Rifles, and in the 1914-1918 war he commanded No. 3 New Zealand Rifle Brigade Field Ambulance. In the war of 1939-1945 he was Regional Deputy in Civil Defence in Auckland.

He held office as President of the Auckland Division of the British Medical Association and of the Pan-Pacific Surgical Association, and was a foundation Fellow of the Royal Australasian College of Surgeons. He was a founder and President of the New Zealand League for the Hard of Hearing.

He will be affectionately remembered by his colleagues in many walks of life, medical, military and civil.

Mr. Neil married Miss Mary Elizabeth Coates, who survives him with four daughters and a son, who practises his father's specialty in Auckland.

Notice.

FAIRFAX READING MEMORIAL PRIZE.

THE Fairfax Reading Memorial Prize of the Dental Alumni Society of the University of Sydney for the year 1954 has been awarded to Dr. J. V. Hall Best. The Fairfax Reading Memorial Prize is awarded biennially and has been awarded on this occasion to Dr. Hall Best for his outstanding services in the organization of the dental profession, in both State and Federal spheres, and for his contributions to the practice of dentistry generally.

Deaths.

THE following deaths have been announced:

LONG.—William Helliar Long, on February 17, 1955, at North Balwyn, Victoria.

BUTLER.—Harry Nairn Butler, on February 22, 1955, at Hobart.

ARNOLD.—Geoffrey Penrose Arnold, on February 28, 1955, at Windsor, New South Wales.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Smith, Herbert Saviour, M.B., Ch.B., 1952 (Univ. Bristol), 7 Tierney Avenue, Matraville, New South Wales.

Ofner, Francis, registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1953, 18 Darley Street, Manly, New South Wales.

Diary for the Month.

MARCH 14-18.—New South Wales Branch, B.M.A.: FOCLA Post-Graduate Course at Wollongong.
MARCH 14.—Victorian Branch, B.M.A.: Finance Subcommittee.
MARCH 15.—New South Wales Branch, B.M.A.: Ethics Committee.
MARCH 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MARCH 16.—Victorian Branch, B.M.A.: Clinical Meeting.
MARCH 17.—Victorian Branch, B.M.A.: Executive of Branch Council.
MARCH 19.—Western Australian Branch, B.M.A.: Annual General Meeting.
MARCH 23.—Victorian Branch, B.M.A.: Branch Council.
MARCH 24.—New South Wales Branch, B.M.A.: Clinical Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 10s. *per annum* within America and foreign countries, payable in advance.